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THE
LARYNGOSCOPE.

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EMBRYONIC HEARING ORGANS AFTER MATERNAL RUBELLA.*†

LEROY A. SCHALL, M.D.; M. H. LURIE, M.D.,
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Since the first communication of Gregg, in 1941, on congenital defects in the progeny after rubella (German measles) of the mother, suffered before or during pregnancy, the question has been discussed with increasing momentum. The present paper is restricted to one detail: deafness in the progeny. As to survey of the literature, at least two competent studies give discussion and extensive bibliographies up to 1949: the thesis of Candiotti, from the Department Portmann, in Bordeaux, and the thesis of Kamerbeek, from the Department Van Gilse, in Leiden.

So far many aspects remain unsettled, even such basic ones as frequency of occurrence. Hiller examined 42 congenitally deaf children in the Tasmanian Institute for the Blind and Deaf. In 32 cases the deafness was due to rubella of the mother in the early months of pregnancy. The remaining 10 were classed as nonrubella inner ear deafness cases, but there

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†From the Department of Otolaryngology, Harvard Medical School and the Department of Otolaryngology, Massachusetts Eye and Ear Infirmary, Boston. Aided by a grant from the Ella Sachs Plotz Foundation.

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was reason to believe that a proportion of these may have resulted similarly from rubella infection of the mother. On the other hand, the Massachusetts survey of Ober, Horton and Feemster found only one case of deafness among the progeny of 54 mothers with rubella during their pregnancies. In this single case the German measles were contracted in the third month.

The situation was summed up by Fowler, in 1950, when he wrote that almost 100 per cent of the offspring have been reported to have congenital defects if rubella was acquired during the middle or first trimester of pregnancy.

The deafness itself is described as of varying degrees up to a complete one with no regular type or curve, even right and left in the same child differing considerably.

In spite of the great amount of published material, pathohistological investigations are conspicuously scarce.

MATERIALS AND METHODS.

The basis of this report is a study of serial sections of three temporal bones originating from two embryos removed by elective abortion after the mothers suffered rubella in the first months of pregnancy. For the autopsy material we are indebted to A. T. Hertig, Professor of Pathology at the Boston Lying-In Hospital.

Case 1: The mother contracted rubella in the eighth week of her pregnancy; in the fourth month the embryo was removed by operation.

One of the temporal bones was secured and after the usual histological preparation sectioned in a roughly horizontal plane. The staining applied was Masson's and a number of sections underwent Bodian impregnation.

The microscopical examination revealed the following: The most conspicuous feature was given by hemorrhages, mostly in the external and the internal ear. In and around the external ear engorgement and extravasates were seen in the temporal muscle, in the skin and in the cartilages of the auricle (see Fig. 1). Hemorrhage had separated the inner lining of the tympanic membrane in circumspect areas, forming cyst-like spaces inside the membrane and between the membrane and the mesenchyma filling the middle ear; in the latter, pneumatization was initiated in several areas.

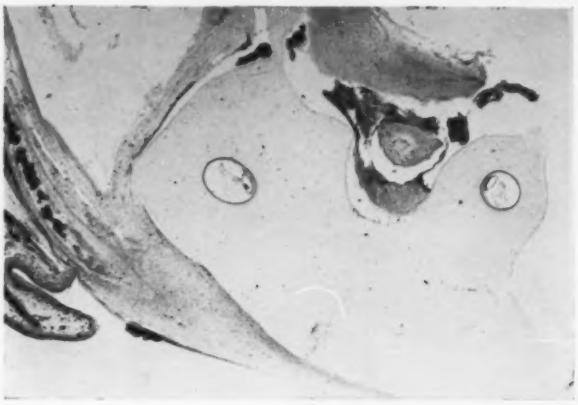

Fig. 1. Massive hemorrhage at the bottom of the subarcuate fossa. Masson, ×17.

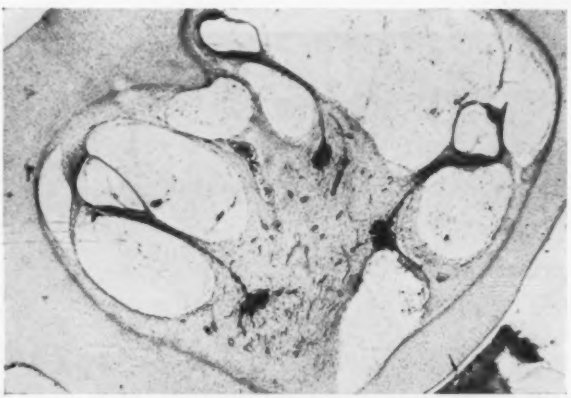

Fig. 2. Cochlear ramuli of the acoustic nerve with spiral ganglion in the connective tissue modiolus of the cartilaginous cochlear capsule. Bodian impregnation, ×28.

Around the inner ear free blood occupied the bottom of the subarcuate fossa (see Fig. 1) as well as the internal acoustic meatus. In the latter, massive bleeding enveloped the cochlear and vestibular branches, while the cochlear branch was damaged by intraneural hemorrhage as well. Inside the cochlea the cochlear artery, in the axis of the connective tissue

Fig. 3. Spiral ganglion and trunks of the cochlear nerve in the connective tissue modiolus of the cartilaginous cochlear capsule. Hematoxylin-eosin, ×28.

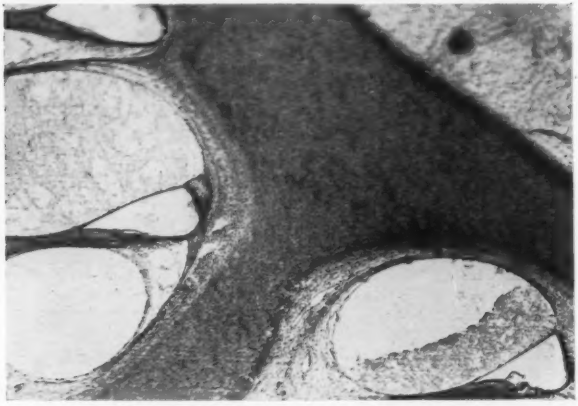

Fig. 4. Corti's organ in the cartilaginous cochlear capsule, beginning to be differentiated in the apical turn; well demarcated in the medial turn; much farther advanced in the basal turn. Hematoxylin-eosin, ×62.

modiolus, was engorged. Blood was seen in all of the three scales. Reissner's membrane was bulging in all turns, with the highest degree of dilatation in the apical turn (see Figs. 4 and 5). Corti's organ was

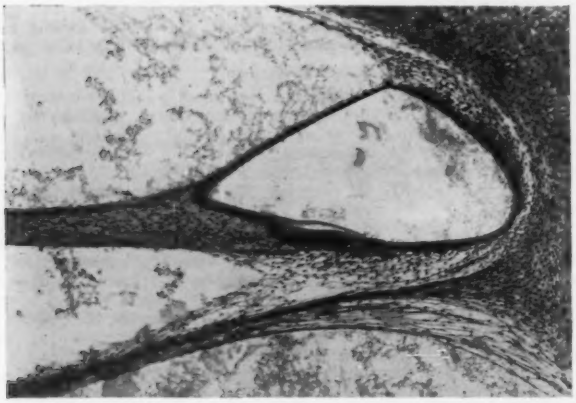

Fig. 5. Corti's organ in an apical turn: flat row of cells giving the impression of a pseudostratified epithelium, covered by the tectorial membrane. Hematoxylin-eosin, ×145.

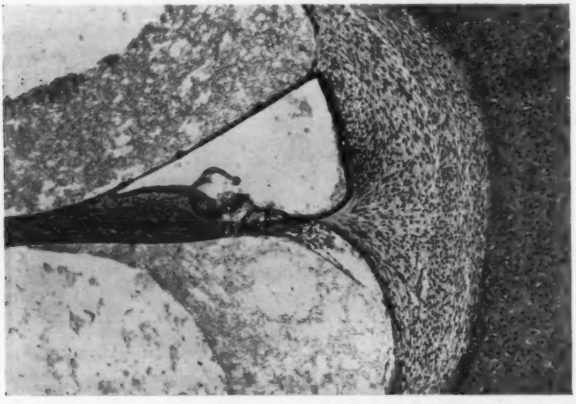

Fig. 6. Corti's organ in a basal turn: loosened cell groups, bulging. Hematoxylin-eosin, ×145.

represented in the middle and in the apical turn (see Figs. 4 and 5) merely by a simple row of cells with the membrana tectoria stretched flatly over them. In the basal turn, Corti's organ was more advanced as to differentiation (see Fig. 6), the flat row being loosened and elevated

into a mound with the tunnel clearly discernible. The semicircular canals showed scattered hemorrhages in the endo- as well as in the perilymphatic spaces, in some points even in the endosteum of a semicircular canal.

Case 2: The 25-year-old primipara contracted rubella six and one-half weeks after conception. As the data of the latter was known, it was possible to compute exactly the age of the embryo, which was removed on the 109th day (at 15 and one-half weeks) by elective abortion. The crown-rump length was 118 mm. Microscopical examination of the eyes (Dr. D. G. Cogan) showed no cataract. The examination of the temporal bones gave the following results:

Right Temporal Bone: Cut in a vertical plane, the pilot series was alternately stained with hematoxylin-eosin and left unstained for observation in polarized light. Engorgement of the soft tissues was conspicuous all over this temporal bone, with hemorrhages scattered about the middle and in the internal ear. In the hypotympanum they appeared in more or less massive form along the walls. They were seen around the crura of the stapes. Between the dura and the labyrinthine capsule, and at the bottom of the subarcuate fossa slight bleedings were found. Pneumatization started in the middle ear, with an open space formed along the tympanic membrane. The vestibular and tympanic scalae were filled with mesenchyma, with the cochlear duct entirely free everywhere. Reissner's membrane was slightly bulging. Corti's organ was represented in the apical turn by a simple row of cells, while in the middle and in the basal turn its development advanced farther, showing a slender mound with the tunnel discernible.

Left Temporal Bone: This was unfortunately removed by a cut which went straight through the cochlea. To avoid further loss of material this accidental plane was maintained under the microtome, with the result of an approximately vertical series of sections. The sections of the pilot series were alternately stained with hematoxylin-eosin and left unstained for observation in polarized light. Beginning pneumatization was seen in the middle ear. In the cochlea, fibrinous masses filled the vestibular and the tympanic scala, while the cochlear duct was free. Corti's organ showed a more progressed stage in the basal than in the middle and in the apical turn. The cochlear aqueduct was filled with a fine meshwork of fibrillary nature. In the semicircular canals, the endolymphatic canals were free while mesenchyma filled the perilymphatic spaces. Opposite the entrance to the modiolus, the acousticofacial ganglionic complex was seen in the adequate stage of development.

COMMENT.

The histological findings have shown features of interest which can be divided into a traumatic and a developmental group.

Two of the temporal bones contained hemorrhages in their different parts besides engorgement of the vessels in the soft parts (see Fig. 1). Seen in the tissues and cavities of the external, middle and internal ear they must be considered a

result of operative trauma. In one temporal bone, engorgement was seen distinctly in the cochlear artery, running in the axis of the connective tissue modiolus.

In the scala vestibuli of an embryo originating from a rubella-mother, Nauta (quoted by van Gilse) saw peculiar blood cell formations of probably pathological significance. van Gilse and Kamerbeek observed hemorrhage in the vestibular scala of a child, deaf from rubella of the mother. De Roetth and Greene reported bloody stools in the newborn child of a mother who had rubella in the fifth week of her pregnancy.

The bleeding tendency in rubella manifests itself in several ways. Wesselhoeft enumerated reports on thrombopenic purpura in connection with rubella. Vascular drainage resulting in thrombotic occlusion through disturbed clotting mechanism was claimed by Putnam to underly encephalitis and meningeal irritation in rubella. Adler's tabulation of 100 encephalitis and encephalomyelitis patients from the Department of Neurology, Harvard Medical School, contained three cases following rubella with no death — and no deafness; they represented "proliferative" encephalitis (proliferation of the glia cells and other tissue elements); the author quoted Ford's case which suggested that toxic as well as proliferative lesions might be present.

It is impossible to decide whether the rubella infection by itself is able to produce hemorrhages, although such a possibility cannot be denied. The difficulty in evaluating hemorrhages may be illustrated by the example of congenital syphilis and by the discussion going on since the publications of Baratoux in 1886-1887, as to whether they are part of the basic pathology or not. Mosher pointed out that hemorrhage in the cochlea is rarely if ever an artefact of a similar character; he wondered whether hemorrhages might not leave behind fibrous tissue in embarrassing locations. Alexander wrote on the danger of excessive scepticism in judging pathological pictures of this kind. The significance of perineural and intraneural interstitial hemorrhages within the internal acoustic meatus and in the intratemporal channels has been

discussed previously; and it was found that cystic "loculaments" of traumatic origin or following hemorrhages in general (see Fig. 1), are sources of cholesteatomas in extrauterine life (Kelemen[1,2]).

In the present specimens, the cochlear aqueduct was closed by a network of mesenchymal tissue, and in this way, even with hemorrhages in the subarcuate fossa and other subdural locations, there was no possibility for invasion of the intracochlear space by this channel.

Hemorrhages as signs of damage done to the vessels (shown in Fig. 1) make one anticipate trouble at the time of the delivery. At this period increased vascular vulnerability will produce pictures which belong to the realm of birth trauma.

The degree of development was found to correspond to the embryonic period. The annulus stapedius, of visceral origin, was firmly fused with the lamina stapedialis, of capsular origin, with beginning differentiation of the annular ligament. The otic capsule presented the homogeneous texture of the original cartilage, with a few areas of ossification disseminated along the semicircular canals. No traces of ossification were found in the connective tissue modiolus; intramembranous bone is first deposited here, according to Bast and Anson, at the fetal age of 20 to 21 weeks. The vestibular end-organs showed the appropriate stage of development. With mesenchyma filling almost completely the vestibular and the tympanic scalae the lumen of the cochlear duct was free; this is in accord with the fact that it differentiates earlier than the perilymphatic scalae.

The organ of Corti followed, in the three turns, the rule that differentiation advances progressively toward the apex (see Figs. 4, 5 and 6). The simple row of cells as seen in the apical turn, reminiscent of pseudostratified epithelium, underwent in the basal turn the characteristic loosening, with appearance of the tunnel and a bulging of the whole cell group, on the way to the definitive formation of the papilla. It is known that an actively differentiating cell is more vulnerable

than a cell in a pre- or in a postdifferentiation stage. The ramuli nervi cochlearis between papilla and spiral ganglion were clearly outlined, with the ganglion formed according to the fetal stage. Among the fibres in the axis, converging to form the trunk of the cochlear nerve, those coming from the basal turn were the most massive (see Figs. 2 and 3).

At this point the findings of Carruthers are of interest. He examined the temporal bones of an infant who died at the age of six and one-half months. The mother suffered from rubella during the first month of pregnancy. The infant had congenital cataracts, patent ductus arteriosus and was thought to be deaf. The semicircular canals, bony and membranous, were well formed. The outstanding feature was the total absence, in both ears, of any differentiation of the primitive cells to form the organ of Corti. The vascular stria contained relatively few vessels, and was shallower and less cellular than usual.

Regarding the mechanism of the effect of a possible toxic agent as to its topographical sequence, observations on animals with spontaneous or experimental cochlear damage give information. In the waltzing guinea pig (Lurie[1]) the degeneration of the organ of Corti, an inherited characteristic, starts in the first and second turns, and progresses towards the helicotrema. It is not a gradual, progressive degeneration, but an irregular one. A great number of the animals examined showed a small remnant of organ of Corti nearly normal at the helicotrema. The original organ of Corti is finally replaced by a simple cuboidal epithelium. The spiral ganglia showed an amount of degeneration which was less than that expected with complete degeneration of the organ of Corti. Nerve fibres were still going to the basilar membrane with no organ of Corti present. This again indicates, as shown in the normal development of the cochlea (see Figs. 2-6), that the organ of Corti is not bound up as intimately with its nerve supply as has been thought.

In animals exposed experimentally to a mechanical or acoustic trauma it was found (Lurie[2]) that disintegration of the cells of the organ of Corti takes place with toxic chemical

changes in the scala media and a possible spreading of the degeneration of the organ of Corti throughout the cochlea; this is a condition progressing for long periods after the actual damage was suffered. In the same species (the guinea pig), the organ of Corti was remarkably resistant amid the changes of spontaneous serous labyrinthitis (Kelemen[3]). Rüedi and Furrer discussed in detail the course of pathological changes in the end-organ and in the nervous apparatus, including their interrelation, in acoustic trauma.

Ingalls, discussing the stage-specificity of this first-trimester infection, mentioned arrested development and vascular anomalies among the possible causes. His Table 1 showed the mean critical period for the development of deafness at 2.17 months of prenatal life. Dietary deficiencies, toxins, duration of stay in oxygen of the premature baby, anoxia by obstetric sedation and anesthesia, may enter the picture in different combinations.

TABLE 1.

SUMMARY OF MICROSCOPICAL FINDINGS IN THE TEMPORAL BONES.

Hemorrhages:

External ear
in skin, temporal muscle, auricular cartilages
engorgement, extravasates
Tympanic membrane
in cyst formation originated by separation of the inner layer

Middle ear
hypotympanum
along walls
around crura of the stapes

Internal ear
outside otic capsule
at bottom of the subarcuate fossa
in internal acoustic meatus
perineural
in cochlear branch
in vestibular branch
intraneural
in cochlear wall
inside otic capsule
in cochlea
cochlear artery, in axis of modiolus, engorged free blood in the three scalae.
semicircular canals
scattered in endo- and perilymph spaces

Developmental Stage:

Mesenchyma

Middle ear
pneumatization starts along tympanic membrane

Internal ear
endolymph spaces free
perilymph spaces filled
cochlear aqueduct filled by fine meshwork

Internal ear
Reissner's membrane
slight bulging in almost every turn
Corti's organ
apical turn: row of cells like pseudostratified epithelium, tectorial membrane stretched out flatly over them
middle turn: in one temporal bone as in apical turn; in the others further advanced in differentiation
basal turn: row of cells loosened, elevated into a mound, tunnel discernible
Nervous apparatus
trunk, spiral ganglion and ramuli of the cochlear nerve in the connective tissue modiolus of the cartilaginous cochlear capsule well developed

Acousticofacial ganglionic complex
in adequate stage of development

Hopkins found no direct correlation between the degree of deafness and the month of maternal rubella, with many cases of profound deafness after infection in the second and third month and with cases of partial deafness in the first month of pregnancy. This contrasted with the experience of Lewis at the Winthrop Foundation at the Massachusetts Eye and Ear Infirmary who found generally a more profound deafness with early maternal infection.

A parallel with the deafness as found by Goodhill in erythroblastosis fetalis shows points of contact as to the possible etiology, as the factor of anoxia, and as to clinical significance. Here as there, among the genetic factors, the father has to be considered. Incidence in the first as against incidence in the subsequent pregnancies has to be evaluated. Audiometric symmetry, both for frequency and intensity, can offer help in finding out about the localization of the damage.

Fox, Krumbiegel and Teresi examined congenital anomalies after maternal measles, mumps and chicken pox. In all cases

of deafness a conduction deafness from otitis media was found, none showing evidence of congenital origin.

Considering the irregularity of findings regarding the hearing of children after rubella of the mother (islands of hearing), the difference in the developmental stage of Corti's organ along the basal lamina is worth watching. The progress between the basal and the apical turns might be interrupted in this period of delicate adjustment, beyond which, according to the general consent, the danger of later deleterious consequences seems to be mostly gone. The lagging behind of one section would produce unpredictable damage to the hearing. A sufficiently large material is needed to obtain information about this important point. van Gilse emphasized that real progress can be expected only when it will be possible to experiment with the pathological agent itself. In all this it has to be considered that, according to Baumgartner, congenital malformation and injury at birth were found, for 1947, among the five leading causes of infant death in the United States.

SUMMARY.

Three temporal bones of two four-month embryos, operatively removed from mothers who suffered rubella in the second month of pregnancy, were histologically examined. Hyperemia and free hemorrhages were seen, while the nervous elements of the inner ear were found to be in a condition corresponding to the fetal age.

This being the period in which the organ of Corti is differentiating along the basilar membrane, any possible disturbance in the delicate mechanism of adjustment, *e.g.*, the lagging behind of certain sections, has to be closely watched as a possible source of hearing defects.

The specific infectious agent causes arrest of development of the end-organ in the most critical period and vascular damage, possibly the latter being the primary change. Vulnerability of the vessels shows up at the end of the pregnancy

in an increased tendency for birth injuries. This way a series of factors collaborate to produce the picture of defective hearing in the progeny, after rubella of the mother.

As far as a survey can be given today, there are two ways in which damage results by exposure to the virus of rubella: one being arrest of development, the other vascular changes. The two can be formulated into a monistic theory by assuming that the vessels are the first to suffer, and lack in nutrition is then followed by developmental arrest. Here is a field of research of rare interest with many problems unsolved. The first decade after the basic discovery of Gregg yielded a rich crop of investigative results, among which, however, pathological findings are negligible. Aycock said that rubella now takes on the status of a disease worthy of the best in diagnosis, treatment, prevention and research. In attacking the problem of the pathology of aural damage it is desirable that the otologist should assume the leading rôle before the initiative is definitely lost to borderline disciplines.

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ACUTE LARYNGOTRACHEOBRONCHITIS. AN ANALYSIS OF 1,175 CASES WITH 98 TRACHEOTOMIES.*†

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There were 1,175 cases of acute obstructive infections of the larynx, trachea and bronchi treated on the croup service at Willard Parker Hospital during the past decade, 1940-1949 inclusive. Of these, 98 (8.3 per cent) were diagnosed as being of diphtheritic origin, and 1,077 (91.6 per cent) nondiphtheritic. At this same hospital the incidence of diphtheria, in a series of 344 croup cases reported by Tolle,[1] in 1929, was over 60 per cent. In 1948, the incidence of croup caused by diphtheria was only 5 per cent (see Table 1). Last year there was only one case.

Of the total 1,175 cases, 32 died, making a total mortality rate of 2.7 per cent. There were 11 diphtheritic croup deaths with a mortality of 11.2 per cent, which was little changed from the 12 per cent reported by Neffson and Wishik in their series of 261 cases treated at Willard Parker Hospital in years 1931-1934;[2] however, there was a dramatic lowering of the mortality rate to 2 per cent in the nondiphtheritic acute obstructive cases as compared with a similar group of 1,379 cases during the preceding decade, 1930-1939, in which there were 136 deaths with a mortality rate of 9.8 per cent (see Table 2).

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*Read at the meeting of the New York Academy of Medicine, Section on Otolaryngology, Nov. 15, 1950.

†Thanks are due Mr. Charles Petersen for helpful assistance in the art work used in this paper.

‡From the Department of Laryngology, Willard Parker Hospital.

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Table 1.

TOTAL OBSTRUCTIVE CROUP CASES AT WILLARD PARKER HOSPITAL
FOR THE 10-YEAR PERIOD 1940-1949

YEAR	CROUP	CASES	DEATHS	% MORTALITY	LARYNGOSCOPED	SUCTIONED	TRACHEOTOMIES	DEATHS	% MORTALITY
1940	DIPHTHERITIC	34	1	2.1	34	34	4	1	25.0
	NON-DIPHTHERITIC	155	0	0.1	137	80	34	5	20.0
1941	DIPHTHERITIC	11	1	9.0	11	9	4	1	25.0
	NON-DIPHTHERITIC	99	3	3.0	87	0	13	2	7.7
1942	DIPHTHERITIC	12	1	8.3	6	4	1	0	0
	NON-DIPHTHERITIC	140	2	1.4	124	16	12	0	0
1943	DIPHTHERITIC	6	1	16.6	5	3	0	0	0
	NON-DIPHTHERITIC	155	2	1.3	78	25	13	2	15.4
1944	DIPHTHERITIC	2	0	0	2	1	0	0	0
	NON-DIPHTHERITIC	92	1	1.1	33	0	6	0	0
1945	DIPHTHERITIC	8	3	37.5	5	5	1	0	0
	NON-DIPHTHERITIC	89	1	1.12	15	9	2	1	50.0
1946	DIPHTHERITIC	15	2	13.3	9	8	0	0	0
	NON-DIPHTHERITIC	71	2	2.8	0	5	3	0	0
1947	DIPHTHERITIC	15	1	6.6	5	4	2	0	0
	NON-DIPHTHERITIC	126	1	.7	9	10	1	0	0
1948	DIPHTHERITIC	13	1	7.7	7	3	0	0	0
	NON-DIPHTHERITIC	77	1	1.3	14	9	4	0	0
1949	DIPHTHERITIC	1	0	0	0	0	0	0	0
	NON-DIPHTHERITIC	63	0	0	4	4	2	0	0
TOTALS	DIPHTHERITIC	98	11	11.2	85	69	13	2	15.4
	NON-DIPHTHERITIC	1077	21	2.0	299	134	86	13	11.4
GRAND TOTALS		1175	32	2.7	384	203	99	15	12.2

A more striking difference is noted in a comparison of two series during the last five years[3] as shown in Table 3. It will be noted that the total mortality rate for these respective periods has dropped from 9 per cent to 1.12 per cent and that the mortality rate in the cases in which tracheotomy was performed was reduced from 28 per cent to 5.5 per cent.

The partial conquest of laryngotracheobronchitis from its original rate of 50 per cent and higher to the present low rate is one of the romances of medicine. The current, almost universal administration of the antibiotics and of the sulfonamides both before and after the patient reaches the hospital is probably the greatest single factor in this favorable trend. The early administration of these agents by the physician in the home has done much to decrease the number of admissions to the hospital and to decrease the severity of the cases that are admitted. Graebner[4] in a review of the records of Willard Parker Hospital for the years 1937 to 1940 indicated this early trend of chemotherapy. He compared a group of

Table 2.

COMPARISON OF FREQUENCY AND MORTALITY FOR SURGICAL INTERVENTION IN NON-DIPHTHERITIC ACUTE OBSTRUCTIVE LARYNGITIS.

	1930-1939	1940-1949
TOTAL CASES	1379	1077
TOTAL MORTALITY, %	9.8	2.0
TRACHEOTOMIES	106	86
TRACHEOTOMY MORTALITY RATE, %	38.6	11.6
FREQUENCY OF TRACHEOTOMIES, %	7.7	8.0
CASES INTUBATED ONLY	130	31
MORTALITY RATE OF INTUBATIONS	46.9	9.7
TRACHEOTOMIES WITHOUT INTUBATION	30	41
TRACHEOTOMIES WITH INTUBATION	76	45
TOTAL CASES INTUBATED	206	76
FREQUENCY OF INTUBATION, %	15.0	7.0
TOTAL TRACHEOTOMIES AND INTUBATIONS	236	117
FREQUENCY OF TRACHEOTOMIES AND INTUBATIONS, %	17.1	10.8
RESULTING PNEUMOTHORAX	13	9
RESULTING PNEUMOTHORAX, %	12.2	10.4

Table 3.

NON-DIPHTHERITIC CROUP CASES AT WILLARD PARKER HOSPITAL. A COMPARISON

YEARS	TOTAL	DEATHS	MORTALITY %	TRACHEOTOMIES	DEATHS	MORTALITY %
1936-1940	616	56	9.0	94	26	28.0
1945-1949	436	5	1.12	18	1	5.5

217 patients admitted to the croup wards in the years 1937 and 1938 receiving no chemotherapy with a similar group of 241 admitted in 1939 and 1940 receiving chemotherapy. The

mortality rate of the later group was approximately one-third less. The major contribution of penicillin is indicated by comparing the three-year period, 1940-1942, when the sulfonamides were widely used with the three-year period, 1946-1948, when both the sulfonamides and penicillin were used. There was a further 50 per cent decrease in the mortality.

That there has been a definite and persistent trend away from mechanical and surgical intervention is clearly illustrated in Table 2. It will be seen that in the last decade the combined frequency of tracheotomy and intubation has decreased from 17.1 per cent to 10.8 per cent. Although the total frequency of tracheotomy increased slightly from 7.7 per cent to 8 per cent, there was more than a 50 per cent decrease in intubations with an increasing number of cases tracheotomized without previous intubation. This is not surprising as the present tendency is to perform a tracheotomy early or not at all. In the entire series, only 43 cases were intubated without subsequent tracheotomy, as seen in Table 4.

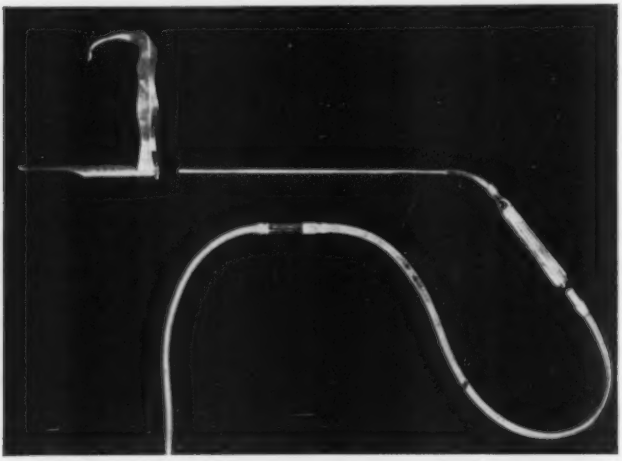
Table 4.

	CROUP	INTUBATION ONLY, NO TRACHEOTOMIES	DEATHS	MORTALITY %
1940	DIPHTHERITIC	3	1	33.3
	NON-DIPHTHERITIC	9	2	22.2
1941	DIPHTHERITIC	1	0	0
	NON-DIPHTHERITIC	8	1	12.5
1942	DIPHTHERITIC	1	0	0
	NON-DIPHTHERITIC	2	0	0
1943	DIPHTHERITIC	2	1	50.0
	NON-DIPHTHERITIC	2	0	0
1944	DIPHTHERITIC	1	0	0
	NON-DIPHTHERITIC	1	0	0
1945	DIPHTHERITIC	0	0	0
	NON-DIPHTHERITIC	0	0	0
1946	DIPHTHERITIC	3	1	33.3
	NON-DIPHTHERITIC	1	0	0
1947	DIPHTHERITIC	0	0	0
	NON-DIPHTHERITIC	5	0	0
1948	DIPHTHERITIC	1	0	0
	NON-DIPHTHERITIC	3	0	0
1949	DIPHTHERITIC	0	0	0
	NON-DIPHTHERITIC	0	0	0
TOTALS	DIPHTHERITIC	12	3	25.0
	NON-DIPHTHERITIC	31	3	9.6
	GRAND TOTALS	43	6	13.9

Consistent with this trend away from surgical intervention is the marked decrease in the incidence of laryngoscopies. In 1940, of 155 nondiphtheritic croup admissions, 137 were laryngoscoped and 60 were suctioned; by contrast, in 1948, of 77 similar admissions, only 14 were laryngoscoped and suction was used in only nine of these (see Table 1).

Various authors contributing to this subject have both written and implied that direct laryngoscopy can now be performed quickly and with such ease and gentleness that it should head the list of routine procedures. There can be no doubt that gentle technique and skill are essential to the successful management of these cases; however, good judgment still is of major importance and gives one a "feel" of the case. This "feel" has been developed to a high degree in some of the nursing supervisors who have been in long and intimate contact with a great number of croup patients. It has been invaluable in helping them sense any change in the patient's condition and in alerting the medical personnel so that appropriate measures could be taken as soon as possible before precious time and the patient's resistance have been sacrificed.

It is difficult to imagine a "completely harmless" laryngoscopy. To subject a severely ill, apprehensive child with a temperature of 104° F. or more to forceful restraint by "mummifying" and to hold the head in such a position as to favor struggle and increased pharyngeal and laryngeal congestion can hardly be considered harmless. In acute laryngeal obstruction the instrumentation may precipitate complete respiratory obstruction; therefore, in the acute emergency, direct laryngoscopy should be attempted only if the diagnosis cannot be established otherwise, or if surgical relief of the obstruction through direct examination and aspiration, intubation or tracheotomy is being considered.[5] Suction equipment and a readily available airway are essential before this procedure should be contemplated. The Willard Parker suction tube devised by Tolle is ideal for this purpose as it both aspirates the trachea and is the best possible airway. This lifesaving tube may be left in place to meet the immediate emergency



and is essential as an airway during tracheotomy. The presence of an airway of this type or a small bronchoscope in the trachea during tracheotomy makes the operation an unhurried, tranquil procedure. It is probably the greatest single factor in lowering the mortality rate of tracheotomy *per se* and in avoiding mediastinal emphysema and pneumothorax.

Sufficient inspection of the throat to rule out diphtheria and retropharyngeal abscess can usually be done easily and quickly by means of a well placed light, a head mirror and a tongue depressor. With the child in as comfortable a position as possible, a careful examination for evidence of diminished breath sounds and areas of congestion should be made. All physical signs of obstruction to the air passages such as suprasternal, supraclavicular, epigastric and intercostal retractions should be carefully appraised and a close watch kept over the patient for any change in these classical and informative signs. Increases of the pulse, respiration and temperature are indications of the severity of the infection but are not useful as indications for tracheotomy; however, fatigue and exhaustion are important and must be carefully evaluated. Tracheotomy should not be delayed if exhaustion is increasing.

Unless tracheotomy is contemplated, it is preferable to keep the child as quiet as possible with steam inhalations, oxygen, or both. The question of steam inhalations versus oxygen remains a controversial one and both have their ardent supporters.

Steam is soothing to the mucous membranes. It helps keep the secretions moist and facilitates expectoration; however, when steam is used, care should be taken to keep the temperature of the room quite low since overheating interferes with the child's rest and is generally debilitating. Mechanical steam or vapor has the desired humidity without the disadvantages of the heat.

Asthma may be overlooked in infants. Allergy plays an important part in a considerable number of cases. Among the types seen in this group are spasmodic croup and allergic edema of the hypopharynx, epiglottis and larynx. Steam usually makes these patients worse. They do much better in a cool oxygen tent. To illustrate this point, it is known that many of these cases improve during the cold ambulance ride to the hospital and they arrive in much better condition than when they left home. Epinephrine solution, 1:1,000, should be given hypodermically, 0.25 cc. to an infant of one year and 0.5 cc. to older children. Epinephrine is well tolerated by children. Whisky, which has often been given to croup cases for its sedative effect, is contraindicated in allergy as it produces vasodilation. An antihistaminic elixir is preferable.

The majority of cases of laryngotracheobronchitis are best treated with a combination of cool vapor and oxygen as advocated by Davison.[6] It has been routine for some time at Willard Parker Hospital to administer penicillin, sulfadiazine and streptomycin to each croup case as soon as possible after admission. The usual dose is 50,000 to 75,000 units of aqueous penicillin (for quick absorption into the blood stream) plus 150,000 to 200,000 units of procaine penicillin in oil, the latter to be repeated every 12 hours. An initial injection of 250 mgm. to 375 mgm. of streptomycin is repeated every six hours. The child is also given 1.5 gr. of sulfadiazine per

pound of body weight every 24 hours, one-half of the 24-hour dose being given initially and then one-sixth of the daily dose every four hours. The oral preparation used is in a liquid form, containing 0.5 gm. of sulfadiazine and 1.5 gm. of sodium lactate in each 5 cc. Since streptomycin is seldom given for a period of longer than one week, no serious toxic symptoms have been encountered. Aureomycin and chloromycetin are also being used at the present time but the results as yet are not conclusive.

There were 98 tracheotomies in this series, 12 in the diphtheritic group and 86 in the nondiphtheritic. There were two deaths in the former group with a mortality rate of 16.6 per cent, and 10 deaths among the nondiphtheritic cases with a mortality rate of 11.6 per cent. The number of cases, deaths and mortality rate for each year is shown in Table 5. The overall mortality rate of 11.6 per cent for 1940-1949 compares very favorably with the 38.6 per cent mortality rate for the 10-year period, 1930-1939.

Table 5.

	CROUP	TRACHEOTOMY WITH OR WITHOUT INTUBATION AFTER ADMISSION	DEATHS	MORTALITY %
1940	DIPHTHERITIC	4	1	25.0
	NON-DIPHTHERITIC	24	10	38.6
1941	DIPHTHERITIC	4	1	25.0
	NON-DIPHTHERITIC	13	1	7.7
1942	DIPHTHERITIC	1	0	0
	NON-DIPHTHERITIC	12	1	8.3
1943	DIPHTHERITIC	0	0	0
	NON-DIPHTHERITIC	13	2	15.4
1944	DIPHTHERITIC	0	0	0
	NON-DIPHTHERITIC	6	0	0
1945	DIPHTHERITIC	1	0	0
	NON-DIPHTHERITIC	7	1	14.2
1946	DIPHTHERITIC	0	0	0
	NON-DIPHTHERITIC	3	0	0
1947	DIPHTHERITIC	2	0	0
	NON-DIPHTHERITIC	1	0	0
1948	DIPHTHERITIC	0	0	0
	NON-DIPHTHERITIC	4	0	0
1949	DIPHTHERITIC	0	0	0
	NON-DIPHTHERITIC	3	0	0
TOTALS	DIPHTHERITIC	12	2	16.6
	NON-DIPHTHERITIC	86	10	11.6
	GRAND TOTALS	98	12	12.2

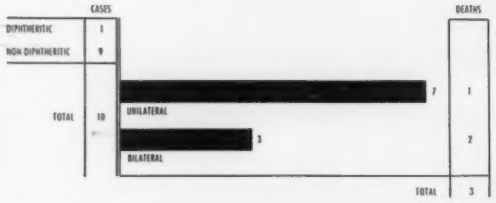
As mentioned above, although there was a slight increase in the frequency of tracheotomy, there was a marked decrease of intubation. In part this may be explained by the tendency to perform the tracheotomy earlier without resorting to intubation and in part to the greater risk and danger from suffocation should the child cough up the intubation tube unexpectedly. With the constant decrease in the number of cases requiring intubation the hospital resident staff is deprived of sufficient opportunity required to attain dependable skill in meeting these emergencies.

In 36 of the patients requiring tracheotomy, the obstruction was due to supraglottic edema, a factor which automatically eliminates the indication for intubation. Slightly more than half of the tracheotomy cases were previously intubated, as shown in Table 4, but at present there is an increasing trend away from intubation.

The incidence of pneumothorax in the total of 98 tracheotomies was 10.4 per cent—nine nondiphtheritic and one diphtheritic (see Table 6). Three were bilateral, two of whom

Table 6.

INCIDENCE OF PNEUMOTHORAX 1940-49



died, and seven were unilateral with one death. Of the unilateral, six were on the left and one on the right side. It is believed that the most important prophylactic measure against pneumothorax is the routine preoperative insertion of an airway, a suction tube, or a small bronchoscope into the trachea. During the operation, then, the child does not struggle against an obstructed larynx, thus drawing in or expelling air from

the chest cavity through the fascial planes of the tracheotomy incision or rupturing the pleura, as a continuous airway is maintained.

The youngest patient in this series was a six weeks old child and the oldest a 53-year-old man. The majority of cases were in the one- to two-year age group. There was a predominance of males (69 per cent) over females (31 per cent), a factor which is quite consistent in most series reported in the literature.

November, December, February and March were the months of greatest incidence.

BACTERIOLOGY.

The bacterial etiology of laryngotracheobronchitis has been a subject of much controversy and will probably remain so for the immediate future. Many authors contend, as Baum does, that the condition is caused by a virus infection, probably influenzal, with a secondary invasion by various pathogens, most frequently the streptococcus.[7] Others contend that most cases of severe supraglottic edema, especially those involving the epiglottis, are caused by the influenza bacillus. Of the cultures taken from the throat, larynx and trachea of our series of 1,077 cases, streptococcus was reported 472 times, staphylococcus 374 times, and an occasional case of influenza bacillus. The influenza bacillus occurred in pure culture in only two cases, both of which were fatal. Their case histories follow:

A three-year-old boy was brought to the "walk-in room" of Willard Parker Hospital by his parents. He was in extreme respiratory distress, and before relief could be given he became cyanotic and expired. Emergency tracheotomy and artificial respiration were immediately carried out, but he failed to respond. The postmortem report in part: "The epiglottis is extremely enlarged and is covered with a purple edematous mucosa; the aryepiglottic folds are also cyanotic and edematous. There is a marked supraglottic edema, the false cords are edematous and congested; the true cords are intact and the ventricles are clear. The subglottic mucosa is only slightly congested and is covered with a minimal amount of mucus. The bronchi contain no mucus. Culture from the larynx was H. influenza and from the blood indicating an H. influenza bacteremia."

The other case was that of a four-year-old white girl with a two-day history of illness. She was brought to the hospital in extreme respiratory distress, marked cyanosis and retractions. She died within five minutes after arrival. An airway tube was passed into the larynx and artificial respiration and epinephrine were given without response. Postmortem examination revealed extreme edema of the epiglottis, aryepiglottic folds and false cords. Cultures from the larynx and also from the blood were positive for B. Pfeiffer.

SUMMARY AND CONCLUSION.

The past decade has seen approximately a 20 per cent decrease in the incidence of these croup cases. The 80 per cent reduction of the overall mortality rate along with the present low mortality of tracheotomies is most gratifying. During this time there has been a marked decrease in the incidence of surgical and mechanical intervention, especially intubation.

There is every reason to anticipate that the future clinical and therapeutic advances will continue toward the ultimate conquest of this vicious disease.

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A THIRD STATISTICAL STUDY OF AUDITORY TESTS IN RELATION TO THE FENESTRATION OPERATION.*

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INTRODUCTION.

This report is a continuation of a long-term study[1-6] of the results of tests of hearing conducted on all of the patients on whom the fenestration operation has been performed by one of us (T.E.W.) at McMillan Hospital (Washington University). It deals with over 100 cases (No. 217 – No. 332 inc.), operated upon for the first time between December 2, 1947 and July 15, 1949. (No "revision" operations are included.) Five cases are arbitrarily omitted: three because of lack of full familiarity with English (their mother-tongue was Spanish) and two with psychological difficulties who did not cooperate fully in some of the tests. The mean age of the group was 32.5 years with a standard deviation (σ) of $\pm$ 8.9 years and a range from 10 to 64 years.

This study is particularly concerned with interrelationships among the speech tests and pure tone tests that were administered to these patients as part of the routine of pre-operative testing and post-operative follow-up.

*This study was begun under Contract N6onr-272 between the Office of Naval Research and Central Institute for the Deaf. It was continued under Contract V1001M-577 between Veterans Administration and Central Institute for the Deaf. The routine testing and the collection of data were later transferred to the Hofheimer Audiology Laboratory at McMillan Hospital (Washington University). The work there is supported by a grant from the Hofheimer Foundation.

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II. PROCEDURE.

The routine of auditory testing was planned to compare:

a) Auditory Test No. 14 (recorded spondaic words, constant level) with Auditory Test No. 9 (recorded spondaic words, diminishing levels),

b) the presentation of spondaic words by "live voice" with the usual (recorded) version of Auditory Test No. 9,

c) "live voice" presentation of the PB word lists with the recorded (Hughes) version of the same lists as a means of determining a patient's maximum articulation score ("PB-max"), and

d) the relationships between speech tests and the pure-tone audiogram in relation to the effects of the fenestration operation.

The tests employed have been described in detail elsewhere.[1,2,3,7]

The routine of testing was as follows:

1) Pure-tone audiometry by air and bone conduction, performed in the Ear, Nose and Throat Clinic at McMillan Hospital according to the usual clinical routine.

2) Speech audiometry was conducted at Central Institute for the Deaf from December 2, 1947 to September 27, 1948. The sequence of tests during this period was:

a) Threshold for speech by Auditory Test No. 9 (spondaic words).[7]

b) Maximum articulation score (PB-max) by recordings of phonetically balanced lists of monosyllables[4] made at Central Institute for the Deaf (voice: R. Hughes). The usual level was 110 db re 0.0002 dyne per cm^2, although for some seriously hard-of-hearing ears 120 db was also used

and for others, following fenestration, 100 db sufficed. If more than one level was used the higher score was taken as the "PB-max". The object of the test was to measure the *maximum* articulation score that the subject could attain.

c) Articulation score (PB lists) at a level 85 db above 0.0002 dyne per cm^2 (PB-85).

First the operated, then the unoperated ear was tested at each step.

On September 27, 1948 the Hofheimer Audiology Laboratory at McMillan Hospital (Washington University) was opened. The routine of speech audiometry in the Hofheimer Laboratory from September 27, 1948 to June 29, 1949 was as follows:

a) Threshold by Test No. 9.

b) Threshold for spondees by live voice (Threshold, L. V.) following the vocabulary and routine of Test No. 9.

c) Maximum Articulation Score (recorded lists) at a level at least 35 db above the Threshold by Test No. 9 if such a level was tolerable, otherwise at the highest tolerable level.

d) Maximum Articulation Score by live voice (PB-max, L.V.) using the PB lists and at the same level as *c*.

e) PB-max (recorded) repeated at the same intensity or, occasionally, a little higher.

About half-way through this series the order of *c* and *d* was reversed. The entire testing session lasted about one and one-half hours.

On June 30, 1949 Auditory Test No. 14 (recorded spondaic words at constant level) was substituted for the measurement of threshold by live voice. The routine was continued without further change until the series was closed on April 1, 1950.

The clinical diagnoses of type of deafness, whether purely conductive, mixed but predominantly conductive, or mixed but mainly perceptive, were made by T.E.W. on the basis of all of the evidence available to him before the operation. All of the fenestration operations were performed with the help of a dissecting microscope. No stopples were employed. At operation T.E.W. measured the length and width of the new fenestra.[6]

III. RELIABILITY AND CORRELATIONS.

1. Threshold Tests: Correlation coefficients and standard errors of measurement.

Descriptions of these statistical measures, with references, have been given in our previous report,[1] and need not be repeated in detail. It will be recalled, however, that the standard error of measurement gives the range above and below the true score within which, for the average subject, a single test may be expected to fall two-thirds of the time on repeated tests.

Test-retest reliability was calculated for the *unoperated* ear as the Pearson product-moment correlation coefficient *(r)* between the pre-operative and the first post-operative test. The coefficients *(r)* obtained in this way from our data are expressions for the stability of these test measures over a period of time. They should thus be distinguished from other expressions for reliability which have been used.[11] (No allowance is made here for learning effects.) A value of r above .80 indicates good reliability and a value above .60 is fair.

The test-retest reliability *(r)* of Test No. 9 (N = 105) is .92 with a standard error of measurement (σ_{meas}) of 2.49 db. There is no significant drift (by the "t" test), such as might be caused by learning, between test and re-test. These values are even better than the values we reported previously[1,3] of r = .89, σ_{meas} = 3.9 db and r = .87, σ_{meas} = 3.1 db respectively.

The test-retest reliability of Test No. 9 presented by "live voice" is even higher. With N = 40, r was .95 and σ_{meas} was 2.60 db.

The estimate of the reliability of gains from fenestration based on Test No. 9 (recorded) alone (calculated by a method similar to that described in reference 9) is .90, while if it is based on the average of Test No. 9 (recorded) and Test No. 9 by "live voice" the reliability rises to an astonishing .99. The number of cases for which the results of both tests are available is small, and in our later comparisons we shall, therefore, reckon the gains from fenestration on the basis of Test No. 9 (recorded) alone. The standard error of the mean gain (σ_m) so calculated is 1.15 db.

The averages of the thresholds (N = 238) obtained by Test No. 9 (recorded) and Test No. 9 ("live voice") are the same within a decibel. The difference is less than the slight systematic error of monitoring (about –1 db) which the tester was found to have developed toward the end of the series. This error fully accounts for the observed difference in the average values.

The agreement between Test No. 9 (recorded) and Test No. 14 (recorded) is practically perfect. The average difference, calculated as No. 9 – No. 14, is .03 db, and r = .96 (N = 72).

2. Maximum Articulation Score: Drift or learning.

The present data allow us to determine whether there is evidence of a drift in the "PB-max" scores from the first to the second test within a single session and also between sessions. In general we find no evidence of any such drift or "learning effect".

Specifically the average differences (pre-operative) were tested by both the "t" and the sign tests. They showed no significant change from the first to the second test. The differences were calculated separately for the cases (N = 59) in which the PB-max was 85 per cent or better (*i.e.* those with good discrimination) and for the cases (N = 33) for which it was below 85 per cent. The averages of the first and of the second tests were within 1 per cent of one another in each case.

The same calculations were made on the differences within each post-operative session. Here there was some slight evidence (by the "t" test) for a poorer performance (by 3.5 per cent) on the second test by the subjects whose PB-max score was below 85 per cent. This questionable downward drift suggests fatigue rather than learning.

The PB-max scores are also reasonably stable from session to session. In the unoperated ear (and omitting all tests made at a level less than 35 db above the threshold for Test No. 9) the average change (for 62 cases with PB-max 85 per cent or higher) is an *improvement* of 1.77 per cent. This is *not* significant by "t" test or sign test. The correlation coefficient *(r)* is + .26. The standard error of measment (σ_{meas}) is 4.54 per cent. (Note that in this series the PB-max is the average of two scores obtained in each session.)

As in the previous series[3] the PB-max scores are less stable when they are below 85 per cent. (The standard error of measurement (σ_{meas}) for the 43 cases with scores below 85 per cent is 6.10 per cent.) The r is higher (+ .53) but there is a slight *deterioration* of PB scores from one session to the next by 2.88 per cent (N = 43). This change is on the borderline of significance (5 per cent level) by the sign test and nearly so by the "t" test. The direction of the change is, however, opposite to the slight upward drift found in our previous series. All in all, therefore, *we have no evidence of a significant systematic drift in the PB-max scores from one session to the next.*

3. *Maximum Articulation Score:* "Live voice" (E.E.) vs recorded (R. Hughes).

There is an average difference of 8.11 per cent (N = 90) in PB-max in favor of "live voice" (Eldert) over the recorded test (Hughes). There is no doubt as to the significance of the difference as the difference was in the same direction *in every case.*

This very considerable and significant difference in articulation scores is in sharp contrast to the very close agreement between "live voice" and recorded test when measuring thresholds. This finding was anticipated but is none the less important. The two voices can be satisfactorily equated by means of the VU meter for measuring thresholds, but they still differ widely in intelligibility when used to measure discrimination, *even though the words and the intensity levels are the same. Articulation scores can only be compared with one another when determined by the same voice,* and the advantage of recorded tests for standardized testing should be obvious.

The lower PB-max scores obtained with the recorded (Hughes) test do not indicate that the recorded version is inferior as test material. The words are a little more difficult to understand; but that is an *advantage,* since it makes the test more sensitive in detecting slight losses of auditory discrimination. E.E.'s voice was actually a little too intelligible. It is difficult for a sympathetic tester not to try to help the patient understand by overenunciating just a little, exactly as we do when conversing with the hard of hearing.

4. Maximum Articulation Scores and the Slope of the Audiogram.

A high-tone hearing loss should make it more difficult for the listener to detect weak fricative consonants and terminal sibilants. To test this assumption we calculated the correlation between PB-max and the slope of the audiogram between 1024 and 2048 cps. The correlation (N = 102) is –.23. This correlation is just barely significant at the 5 per cent level.

A far more significant correlation was found between PB-max and the slope of the audiogram between 1024 and 2896 cps. This correlation (N = 102) is –.43, and is significant at the 1 per cent level. In both cases the greater high-tone loss is associated with lower PB-max scores.

Likewise the PB-max correlates significantly (1 per cent level) when the tri-serial eta is calculated ($\eta = .31$) for the means of the followings groups:

	Mean PB-max score
Pure Conductive (N = 76)	= 90.0%
Conductive-mixed (N = 23)	= 86.1%
Mixed-nerve (N = 3)	= 80.7%

5. Maximum Articulation Score and Intensity Level.

PB-max (recorded, Hughes) was plotted against the number of decibels above the No. 9 threshold at which the test was administered. There is a large spread of values, but a slight trend is evident when the average values are plotted for three ranges of intensity, as follows:

	Mean PB-max score
35-39 db re No. 9	= 85.0%
40-44 db re No. 9	= 88.7%
45 and over db re No. 9	= 90.2%

The shape of the average articulation curve for the entire group will be considered in a later communication.

6. Reliability of Articulation Score at 85 db (PB-85).

Part of the data employed in the foregoing sections was obtained from the articulation tests that were administered at the arbitrary level of 85 db re 0.0002 dynes per cm^2 (corresponding to very loud speech). The test-retest reliability of the tests at this level (only 17 cases available with two such tests) is $r = .68$. The standard error of measurement is 7.83 per cent. These figures compare with $r = .55$ and $\sigma_{meas} = 4.23$ per cent for the PB-max of the same 17 cases. The higher r and σ_{meas} are to be expected because of the greater scatter when the articulation score is measured on the steep part of the articulation curve below the plateau.[8]

IV. GAINS FOLLOWING FENESTRATION.

The gains in decibels following the fenestration operation have been analyzed in full elsewhere,[5] but several correlations among the gains measured in various ways are of some interest.

1. Relation to Size of Fenestra.

Measurements were made at operation of the size of the fenestra produced in the horizontal semicircular canal, and in general the surgeon attempted to make as large an opening as seemed practical. The reason for this was to reduce the likelihood of closure. The incidence of closure has been analyzed elsewhere;[6] but we have also examined our data to determine whether there is any relation between the size of the fenestra and the resulting improvement in the threshold for speech.

Correlations were calculated between the improvement in the threshold for speech and *a)* the width of the opening, *b)* the length of the opening, and *c)* the area of the opening (proportional to the product of length by width). The first was calculated as tri-serial eta ($\eta = .23$) and the two latter as product moments ($r = -.05$ and $r = .08$ respectively). The number of cases (N) was 99 in each case. None of the correlations is statistically significant. This negative result is not surprising as the variations in size were not great, and particularly as we have shown elsewhere that the gain for speech depends primarily on the amount of conductive deafness that the patient happens to have at the time of the operation.

Inasmuch as the most predictable result of the fenestration operation is not the *improvement* in the threshold for speech but the *absolute level* of the (post-operative) threshold[5] it seemed possible that some relation between the immediate post-operative threshold and the size of the fenestra might be found. The calculations were based only on the cases diagnosed as pure conduction deafness in order to avoid contamination by nerve deafness, which is not affected by the opera-

tion. Cases in which some "surgical accident" occurred were also omitted. The calculations (tri-serial eta and analysis of variance) showed *no* significant correlation between the post-operative thresholds and either the width, the length or the area of the fenestras as measured by the surgeon. It should be noted, however, that the fenestras were all between 1.0 and 1.5 mm. in width and between 2.5 and 4.0 mm. in length.

2. Gain for Speech vs Gain by Air Audiogram.

The correlation between the immediate gain for speech (Test No. 9, measured approximately three months after operation) and the gain for pure tones (average for 512, 1024, 2048 and 2896 cps, measured at the same sessions as the speech thresholds) is $r = +.74$ ($N = 103$). This is exactly the same coefficient as we found in both our first and our second series.[1,3]

The mean gain by Test No. 9 in the present series is 23.52 db; by air audiogram it is 19.98 db. As before, the gain measured by Test No. 9 is significantly larger (beyond the 1 per cent level of confidence by the "t" test) than when it is measured by averaging the pure-tone audiogram over the speech range. The discrepancy of 3.54 db in favor of the direct measurement is practically identical with the similar discrepancies we found in our two previous series (4.06 and 3.40 db respectively).[1,3]

3. Gains for Different Frequencies of Pure Tone.

As before, the gains at 512 and 1024 cps are practically the same as for Test No. 9 while the gains for 2048 and 2896 cps are significantly less.

The mean gains at the various frequencies ($N = 106$) are:

512 cps = 22.45 db
1024 cps = 23.25 db
2048 cps = 20.24 db
2896 cps = 11.60 db

The only differences that are not significant are those between 512 and 1024 and between 512 and 2048 cps. This finding confirms our previous results.

It seemed possible that the smaller gains at 2048 and 2896 cps might be only a temporary deficit. We therefore compared the average gains for the air audiograms as a whole and for 2048 cps separately as measured 6 to 15 months after operation. The gains were not significantly greater than the immediate gains of the same group of patients (N = 40). Neither did the gains measured 12 months or more after operation differ significantly from the results of the "immediate" tests (N = 12, sign test). Likewise the relation between the gain for speech (Test No. 9) and the gain by average air audiogram showed *no* significant difference after 11 months from the relations calculated from the immediate gains and given in sub-section 2 above. (N = 66; "t" and sign tests). We conclude that the gains measured within the first three months remain substantially stable on the average during the following year.

We still have no explanation to offer for the relatively smaller gains at the higher frequencies following fenestration.

4. Prediction of Gain for Speech.

We have shown in a previous paper[5] that the gain for speech following fenestration depends primarily, as we should expect, on the amount of conductive loss present before the operation. To test this conclusion further we have calculated the correlation between gain for speech (Test No. 9) and the pre-operative conductive loss. The measure of conductive loss was the pre-operative difference between the loss by air conduction and the loss by bone conduction (average of 512, 1024, 2048 and 2896 cps). The value of r (N = 50) is + .45. This is a statistically significant correlation and confirms the concept elaborated in our previous paper.

5. The Effect of Fenestration on Maximum Articulation Score.

The fenestration operation did not significantly alter the maximum articulation scores of the operated ears (N = 103). The observed differences were *not* significantly different by either "t" test or sign test, not even for the sub-group (N = 23) whose pre-operative tests were performed at levels slightly less than 35 db above their No. 9 thresholds. This constancy of the maximum articulation score was expected theoretically and agrees with our previous results.[3]

Incidentally, the average PB-max (for cases tested 35 db or more above the threshold for Test No. 9) of the *operated* ears was compared with the corresponding average PB-max of the *unoperated* ears. The difference of 1.75 per cent in favor of the latter is *not* significant.

DISCUSSION.

This statistical study of the auditory tests administered to approximately 100 additional patients on whom the fenestration operation has been performed has yielded little that is new but it confirms quantitatively as well as qualitatively many of the conclusions derived from our previous studies. In no case where the same points were re-examined has it been necessary to modify significantly our earlier conclusions. In view of this stability of the results it should not be necessary to repeat these particular studies until some significant change in technique is introduced, either in the operation itself or in the tests of auditory function.

In addition to the data here reported we have also analyzed with some care the articulation scores obtained with our recorded versions of the PB lists. The results of this study will be reported in the near future.

SUMMARY.

Speech tests and pure tone tests of hearing were administered to over 100 nearly consecutive patients before and after

the fenestration operation. The reliability, the standard error of measurement, and other statistical indices were calculated. The results agree very closely with those of our previous similar studies.

The spondaic word tests are highly reliable as tests for the threshold for speech. The two recorded versions, Test No. 9 (descending levels) and No. 14 (constant level), and administration by "live voice" are all practically interchangeable.

The maximum articulation score (PB lists given at least 35 db above the threshold for Test No. 9) is significantly higher when the lists are given by "live voice".

The maximum articulation scores ("PB-max") show no evidence of a learning factor from one session to the next under the conditions of our clinical routine testing.

The maximum articulation score is slightly but significantly lower for the patients with some high-tone nerve loss.

The articulation score at the arbitrary level of loud speech, i.e. 85 db re 0.0002 microbar, is a less stable measure than the maximum articulation score.

No significant correlation was found between the size of the fenestra (measured at operation) and either the threshold for speech or the gain for speech following the operation.

As in our previous studies the average gain from fenestration is less for frequencies 2048 and 2896 cps than for frequencies 512 and 1024. The gains at the lower frequencies agree very closely with the gain measured by Auditory Test No. 9. The relative deficit at the higher frequencies is still present after one year and is probably permanent.

The fenestration operation does not significantly alter the maximum articulation score of either ear.

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HEADACHE AND VERTIGO ASSOCIATED WITH HYPOGLYCEMIC TENDENCY.*†

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Considerable detailed information regarding the various aspects of spontaneous hypoglycemia is contained in the medical literature since Harris,[1] in 1924, described a fairly characteristic group of symptoms experienced by individuals with abnormally low blood sugar levels. Little or no attention has been placed on an even larger group of patients whose symptoms are directly related to lowered blood sugar levels. Headache and, to a lesser extent, vertigo are common symptoms experienced by this latter group. In practically every case the headache and vertigo will be reproduced during the course of the five-hour glucose tolerance test.

The etiologic classification of spontaneous hypoglycemia as set forth by Conn[2] applies to this group of patients. The available information placed these patients in the functional or organic hepatogenic groups.

ETIOLOGIC CLASSIFICATION OF SPONTANEOUS HYPOGLYCEMIA.

I. Organic—recognizable anatomic lesion.

A. Hyperinsulinism.

1. Pancreatic island cell adenoma.

a. Single.

b. Multiple.

c. Aberrant.

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2. Pancreatic island cell carcinoma.

   a. Localized.
   b. With metastases.

3. Generalized hypertrophy and hyperplasia of the islands of Langerhans.

B. Hepatic disease.

1. Ascending infectious cholangiolitis.
2. Toxic hepatitis.
3. Diffuse carcinomatosis.
4. Fatty degeneration or "fatty metamorphosis."
5. Glycogenosis (von Gierke's disease).

C. Pituitary hypofunction (anterior lobe).

1. Destructive lesions (chromophobe tumors, cysts).
2. Atrophy and degeneration (Simmond's disease).
3. Thyroid hypofunction (? secondary to pituitary hypofunction).

D. Adrenal hypofunction (cortex).

1. Idiopathic cortical atrophy.
2. Destructive infectious granulomas.
3. Destructive neoplasms.

E. Central nervous system lesions (Hypothalamus or brain stem interference with nervous control of blood sugar).

II. Functional—no recognized anatomic lesion but explainable on basis of unusual somatic function.

A. Hyperinsulinism (imbalance of the autonomic nervous system); hypoglycemic fatigue; nervous hypoglycemia; functional hypoglycemia; reactive hypoglycemia.

B. Alimentary hyperinsulinism (rapid intestinal absorption).

1. After gastroenterostomy.

2. After gastric resection (partial or total).

C. Renal glycosuria (severe degrees of low renal threshold for dextrose).

D. Lactation.

E. Severe continuous muscular work.

III. Miscellaneous.

A. Factitious (surreptitious insulin administration).

B. Postoperative hypoglycemia.

C. Severe inanition.

D. Unknown.

In order to associate some relationship between the patient's symptoms and the blood sugar levels, the standard preparatory diet was not used, and the patient was instructed to remain on his usual diet until after the glucose tolerance test was made.

The nature of the diet preceding the test has a marked influence on the results of the test in both animal and man. The restriction of carbohydrates in man results in a marked decrease in ability to utilize carbohydrates and produces an exaggerated glucose tolerance curve. A diet high in carbohydrates, low in protein, produces an increased tolerance and the curves are low and flat. A diet high in fats produces a much greater and more prolonged hyperglycemia when glucose is given than does one high in carbohydrates. The ingestion of large amounts of protein is followed by very little rise in the blood sugar. The long period required for the absorption and conversion of protein gives a steady supply of dextrose over a fairly long period of time, resulting in very little elevation and secondary fall of blood sugar levels. These facts account for the therapeutic effectiveness of a high protein diet in cases of functional hypoglycemia.

The effect of high and low carbohydrate diets preceding the oral administration of glucose is shown in Fig. 1.[3]

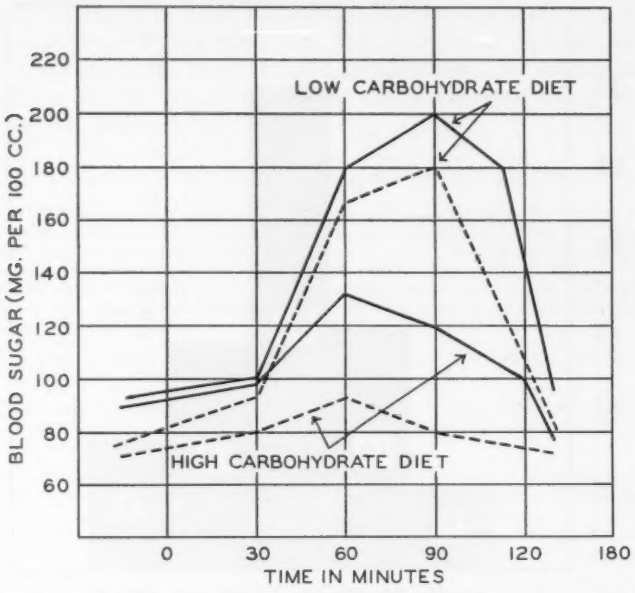

Fig. 1. The influence of high and low carbohydrate diets preceding the oral administration of 50 gm. of glucose ————, capillary blood sugar; - - - - -, venous blood sugar. (Courtesy, Kolmer: Clinical Diagnosis by Laboratory Examinations, 2nd ed. New York, N. Y.: Appleton-Century-Crofts, Inc.)

The case histories of 72 patients complaining of headache or vertigo and whose symptoms were relieved by high protein diet were reviewed. In all patients the symptoms were reproduced or aggravated during the five-hour glucose tolerance test.

Forty-five (62.5 per cent) were females. Thirty (41.8 per cent) of the patients were in the third decade (see Fig. 2). Headache was the chief complaint of 70; vertigo, in two. Both vertigo and headache were present in 32 (44 per cent).

The headache was described as being dull in 65 of 65 cases and throbbing in 56 of 59. It was relieved by pressure in 18

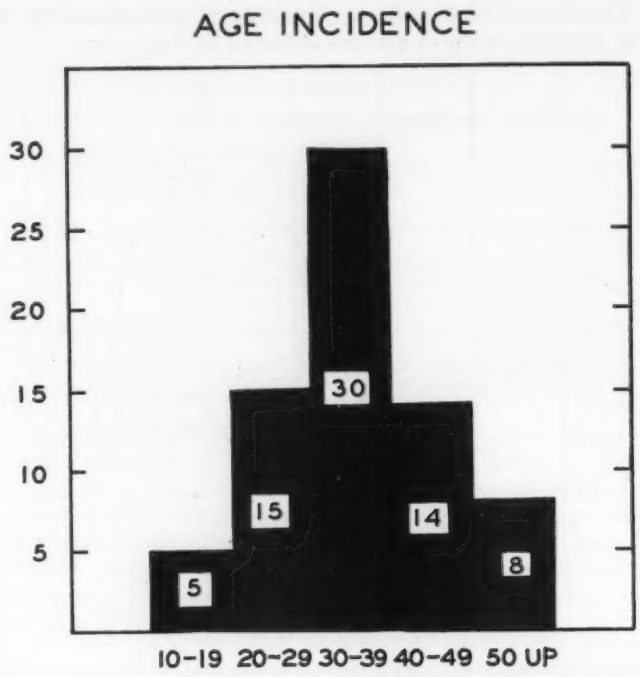

Fig. 2. Age incidence.

of 32 cases. Forty-two of 56 patients were awakened at night by headache, and in 44 patients, 39 had headache before breakfast. In 33 of 48 patients the headache was definitely related to meals.

The headache experienced is visceral in character and is described as being dull, throbbing and is often relieved by pressure. This is the same type of pain that was produced experimentally by Ray and Wolff[4] by mechanical distention of the superficial temporal artery. The same type of pain is experienced when a hypodermic needle is pushed through the wall of an artery or vein.

The glucose tolerance curve, in most instances, was low, especially from the second through the fifth hour. In only 15 cases did the fifth hour level reach the fasting level, and in seven of these 15 the curve was flat. Symptoms were usually produced when the level dropped to 70 or less. In others, the rapidity of the fall of the sugar levels appeared to be the important factor.

The following abbreviated case histories are typical:

Case 1: Mrs. D. B. H., age 32 years, was first seen on Aug. 21, 1947, with the chief complaint of vertigo of three months' duration, which was associated with some nausea and seemed to be more or less continuous in character and increased in intensity at times. The patient stated that she was subject to motion sickness. Vertigo, nausea and jitteriness were present if a meal was missed. There was no history of headache, but occasionally a sensation of pressure was present in the vertex.

The past medical history was noncontributory. The patient's mother had asthma for a number of years.

The ear, nose and throat examination did not reveal any gross deviation from the normal. A medical and ophthalmological examination revealed nothing significant.

The five-hour glucose tolerance test gave the following results: Fasting—80 mg. per cent; one-half hour—125 mg. per cent; one hour—104 mg. per cent; two hours—73 mg. per cent; three hours—70 mg. per cent; four hours—58 mg. per cent; five hours—59 mg. per cent. During the glucose tolerance test, the patient experienced a considerable amount of vertigo associated with headache.

The patient was given a high protein, low carbohydrate diet and was instructed to eat between meals and before bed time. Within a very short time, all symptoms had disappeared and she had remained free from attacks when last seen on Jan. 5, 1949, unless she had changed her diet.

Case 2: Mrs. E. F. D., age 35 years, was first seen on Nov. 8, 1948, complaining of occipital and left temporal headache which was dull and throbbing in character and not relieved by pressure. The patient's symptoms had been present for four years but had become more severe for the last years. She was occasionally awakened at night with headache, and it was usually present in the morning before breakfast. Occasional vertigo was associated with the headaches. The patient was quiet fond of soft drinks and consumed a number each day.

The past medical history was noncontributory except for symptoms suggestive of hay fever.

The ear, nose and throat examination was essentially negative except for some slight polypoid changes on the posterior tips of the inferior turbinates and the presence of a small polyp which was removed from the left middle meatus.

Medical examination failed to reveal abnormalities which could be associated with her headaches.

The five-hour glucose tolerance test revealed the following: Fasting—89 mg. per cent; one-half hour—115 mg. per cent; one hour—102 mg. per cent; two hours—103 mg. per cent; three hours—91 mg. per cent; four hours—68 mg. per cent; five hours—68 mg. per cent. During the glucose tolerance test, the patient experienced headache similar in character to that which she had been having for the past four years.

A diet high in protein and low in carbohydrates was prescribed and her symptoms gradually disappeared and when last seen on May 2, 1949, she was symptom free.

Case 3: Mrs. M. E. G., age 37 years, was first seen on Jan. 7, 1949, complaining of dull, throbbing pain in and about the left cheek and eye for approximately one year. The patient was overweight and had been on a strict diet for some time.

A medical examination and neurological consultation revealed nothing eventful.

The five-hour glucose tolerance test revealed the following: Fasting—103 mg. per cent; one-half hour—198 mg. per cent; one hour—222 mg. per cent; two hours—193 mg. per cent; three hours—140 mg. per cent; four hours—69 mg. per cent; five hours—65 mg. per cent. During the fourth and fifth hours of the glucose tolerance test, the patient experienced pain similar to that which she had had for the past year.

The patient was placed on a high protein and low carbohydrate diet. Her symptoms quickly disappeared and when last seen on May 3, 1949, she was completely free of her symptoms.

Case 4: Miss P. A. H., age 31 years, was first seen on July 4, 1949, complaining of frontal headache which was dull, throbbing in character and was relieved by pressure. The patient was often awakened with headache between 12:00 and 2:00 o'clock A. M. The patient experienced a similar type headache when a meal was missed and some jitteriness was associated with the headache. The patient had been on a diet low in protein.

The ear, nose and throat examination was essentially negative and medical examination was noncontributory.

The five-hour glucose tolerance test revealed the following: Fasting—66 mg. per cent; one-half hour—98 mg. per cent; one hour—125 mg. per cent; two hours—89 mg. per cent; three hours—67 mg. per cent; four hours—41 mg. per cent; five hours—64 mg. per cent. During the five-hour glucose tolerance test, the patient experienced similar headache and was quite nervous and felt faint throughout the duration of the test.

She was placed on a high protein, low carbohydrate diet with between meal feedings. Marked improvement was noted in a short time and when last seen in November, 1950, she was having occasional difficulty.

SUMMARY.

1. Headache and vertigo are common symptoms associated with lowered blood sugar levels.

2. The headache is described as being dull and throbbing in character. It is often present at night and may be related to meals.

3. Patients in the functional group respond well to a diet high in protein and low in carbohydrates. Supplementary feedings may be necessary.

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MORE COMPLETE ADENOIDECTOMY TO OVERCOME INTERFERENCE WITH NASAL BREATHING BY THE SALPINGONASAL AND SALPINGOPALATINE FOLDS.*

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Most experienced otolaryngologists consider tonsillectomy in children as a relatively simple procedure, but the adenoidectomy and concluding hemostasis as posing a more difficult problem. During the past 10 years, one of us (E. R.) has been using the modified Coakley[1] slip knot tie to control the bleeding. At first only the bleeding vessels in the lower part of the wound were tied; but after skill had been acquired, the source of bleeding higher up was investigated.[2]

In the course of searching for these vessels, it was noted that sometimes, even after what was considered a complete adenoidectomy with LaForce adenotome and curette, there remained, anterior to the pharyngeal tonsil, a thick rugged mass which could not be removed with these instruments without endangering the tube.

The pharyngeal tonsil is defined by Shambaugh in Jackson's book[3] as being "a prominence on the roof and posterior wall of the nasopharynx produced by lymphoid tissue," but this previously mentioned rugged fold was not connected with the pharyngeal tonsil, although it had the appearance of adenoid tissue. It was not located on the posterior wall of the pharynx but was primarily on the palate. The fold was anterior to a catheter inserted into the ostium of the Eustachian tube. The

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finding of the plica salpingopharyngea directly behind the inserted catheter made it clear that this rugged fold was the plica salpingopalatina.

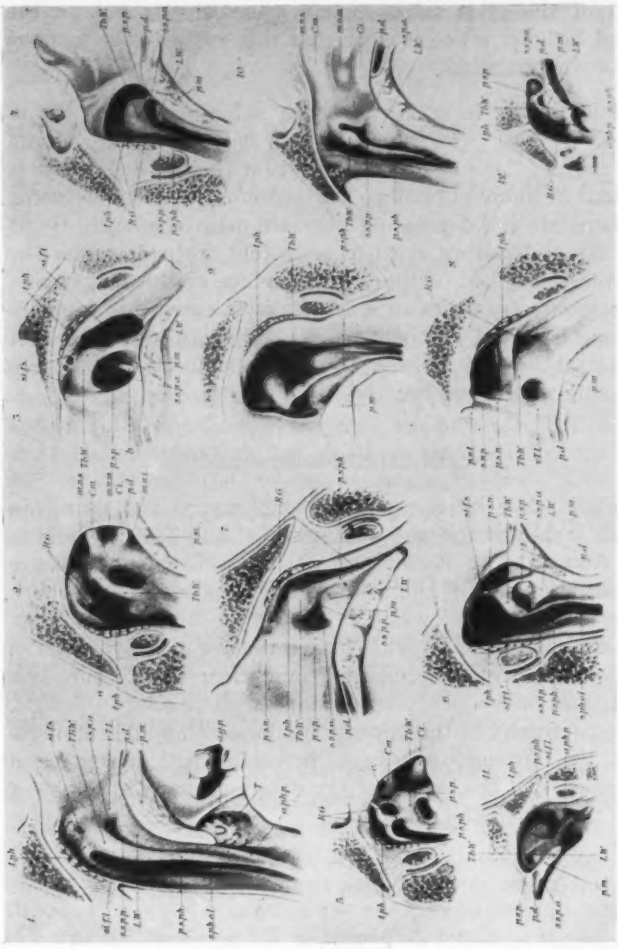
Because of its location, the plica salpingopalatina is not obvious. In most cases it can be seen only during anesthesia in the course of the operation. The best time to look for it is after tonsillectomy but before adenoidectomy. After elevating the soft palate and depressing the main mass of adenoid tissue in the dorsal direction, one can see a fold protruding more or less prominently out of the dorsal surface of the soft palate, quite separate from the adenoid. In most cases this protruding tissue is only 1 or 2 mm. wide. But in 10 of our cases it was at least 9 mm. wide, obstructing one or both choanae and interfering with nasal breathing.

ANATOMICAL CONSIDERATIONS.

Excellent descriptions of the epipharynx are found in Luschka's[4] monograph and in Barnhill's[5] and Testut's[6] anatomies. According to Escat[7] and St. Clair Thomson's[8] lifesize drawings, the height and width of a choana of a newborn averages 6.5 mm. The width in a five-year-old child is 9 mm. and in an adult 13 mm. This means that in a five-year-old, a salpingonasal or salpingopalatine fold wider than 6 mm. is a serious, and one wider than 9 mm. is an absolute obstacle. With hypertrophy of the plicae salpingopalatina and salpingonasalis, the obstruction to nasal breathing will depend upon the location and the extent of the enlargement of the fold as well as the size of the choanae.

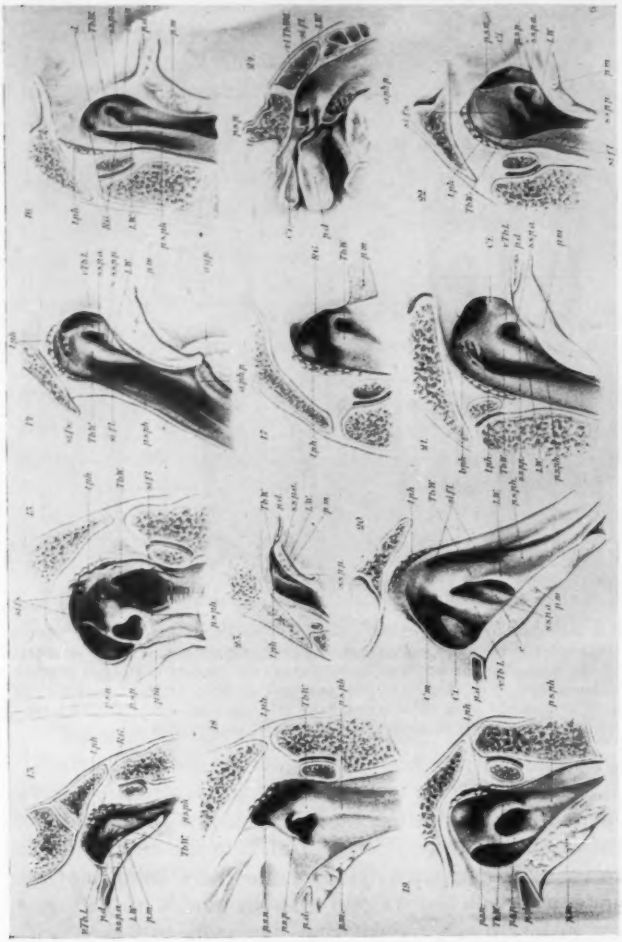
The following lines are excerpts translated from a very accurate anatomical description of the rhinopharynx by Kostanecki:[9]

"The appearance of the anterior lip of the tubal ostium varies greatly in the different specimens (see reproduction. In Figs. 1, 2 and 8 it is small; in Figs. 3, 4, 5, 6, 7, 14, 15, 16 and 17 it is larger.). These variations are caused by developmental differences in strength of a ligament which can be



(a.g.p.) arcus palatoglossus; (a.ph.p.) arcus pharyngopalatinus; (b.ph.) bursa pharyngea; (m.n.i.) meatus nasium inferior; (m.n.m.) meatus nasium medius; (m.n.s.) meatus nasium superior; (c.i.) concha inferior; (c.m.) concha medium; (c.s.) concha superior; (p.d.) palatum durum; (p.m.) palatum molle; (p.s.n.) plica salpingonasalis; (p.s.p.) plica salpingopalatina; (p.n.l.) plica nasalis lateralis; (a.ph.) plica

found to be a continuation of the membranous tube into the soft palate. This ligament was named by Tourtual,[10] the anterior salpingopalatine ligament. The shape of this liga-



salpingopharyngea; (R.G.) fossa of Rosenmüller; (si.f.l.) sinus fauclum laterallis; (si.f.s.) sinus fauclum superior; (si.sph.) sinus sphenoidalis; (si.n.p.) sinus nasalis posterior; (si.ph.o.l.) sinus pharyngo-oralis lateralis; (s.s.p.a.) sulcus palatinus anterior; (s.s.p.p.) sulcus palatinus post.; (T) tonsil; (Tb.W.) Torus tubarius; (t.ph.) tonsilla pharyngea; (V.T.L.) anterior lip of the tube; (L.W.) bulge of the levator.

ment varies very much in different anatomical specimens. Sometimes it is composed of well developed, tendon-like strands; at other times it is only a fine fibrous continuation

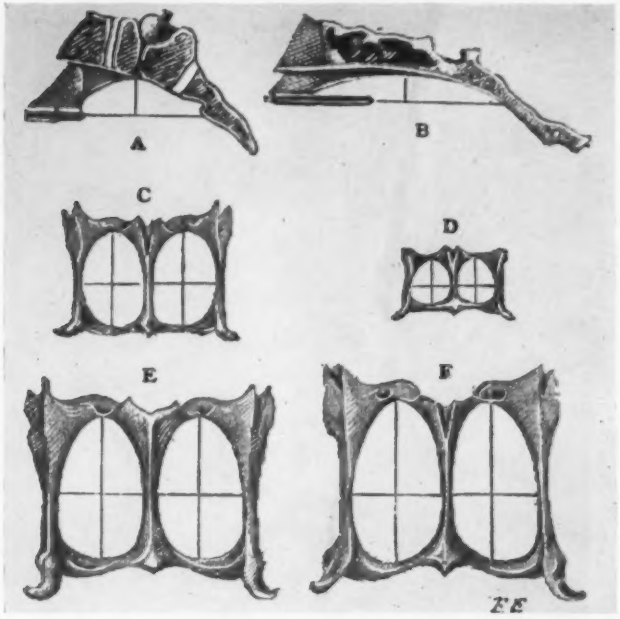


Fig. 176. Lifesize drawings of the nasopharynx. From Sir St. Clair Thomson's Diseases of the Nose and Throat (3rd ed., 1926), p. 333. (A) Sagittal section of the nasopharynx at birth, showing persistence of the canal lodging Rathke's pouch (cf. Fig. 2, p. 2); (B) sagittal section of the nasopharynx of a dog; (C) choanae of a child aged five; (D) choanae at birth; (E) choanae of an adult female; (F) choanae of an adult male., (Escat.)

of the membranous tube, originating from the lateral part of the tubal cartilage. Sometimes it extends to the midline of the hard palate or it may turn dorsally into the substance of the soft palate, where its fibres blend with the tendon of the tensor veli, the posterior salpingopalatine ligament, and even with the salpingopharyngeal ligament. The result is that it curves about the tubal ostium like an arc.

"The mucosa of the anterior lip of the tube is very stiffly connected with these fibrous bands. Tourtual[10] found that the

anterior lip of the tubal ostium is always accentuated by a strong fold of mucosa which he named the salpingopalatine fold. The appearance of this fold depends upon the developmental differences in the strength of the underlying ligament and the thickness of the mucosa covering it. It may be thin and transparent, if the layers of glands and lymph follicles are thin; or it may be thick and very prominent. In its downward course, the salpingopalatine fold runs either directly to the midline of the palate or it describes an arc, the concavity of which faces the ostium.

"Besides this fold which is directed downward, there is another very important fold of the mucosa which originates on the upper part of the tubal torus and is directed upward. This fold was named by Hoffman, the 'salpingonasal fold.' Its thickness is variable. Under the mucosa of the salpingonasal fold he found a continuation of the basilar fibrocartilage composed of small but strong fibres which are strongly blended with the perichondrium. Often the fibres of the anterior palatine ligament unite with these fibres.

"For this reason, the salpingonasal and salpingopalatine folds are often found forming one continuous fold instead of two separate ones (see reproduction Figs. 6, 7, 8, 15, 18 and 19).

"Anterior to the salpingonasal fold, another fold, called by Tourtual the lateral nasal fold, was often found. This continues downward anteriorly to the plica salpingopalatina, so that one sees what appears to be a double salpingopalatine fold (see reproduction Fig. 8). The anterior lip of the tube and the folds originating from it greatly influence the size and form of the choanae."

In our experience, the salpingopalatine fold was always visible. In most cases it originated from the lateral lamina of the tubal torus and formed an arc with the concavity facing the tube. It ended mostly on the dorsal surface of the palatopharyngeal fold. Often the fold did not start at the tube but

seemed to originate from the fornix; that is, the salpingonasal and salpingopalatine folds united to make one continuous arc.

PATHOLOGICAL SIGNIFICANCE OF THE HYPERTROPHY OF THE FOLDS.

The importance of the nasopharynx is well emphasized in the excellent symposiums of LeJeune[11] and Dudley.[12] Hollender[13,14] reports the nasopharyngeal findings in a group of 131, and then 140, autopsies. Lyons,[15] Baker,[16] Giddal,[17] Bordley[18] and Dudley[19] write imposing articles confirming the vital importance of the nasopharynx as a source of focal infection, deafness and mouth breathing.

In a brilliant article, Meltzer[20] expresses the opinion that many an irradiation will not be necessary if the lymphoid tissue is removed completely from the nasopharynx.

Our material comprised 155 children ranging from two to 10 years in age. All operations were deferred until at least two weeks after the last acute infection. The cases were not selected at random; many of them were referred by doctors who knew that one of us was trying to resolve these obstructions to nasal breathing by operation.

Thirty-eight cases showed marked hypertrophy of the salpingonasal and salpingopalatine folds. In several cases, the plicae were 10 mm. wide; in one case, 12 mm.; and in one striking instance, 15 mm. wide in its upper part and 10 mm. wide in its lower part.

This last case (No. 103) was that of an eight and one-half year old child whose tonsils and adenoids had been removed six years before. As far as the mother remembered, the child had been a mouth breather all his life. The operation, although performed by a very reliable surgeon, did not relieve the obstruction to the nasal breathing. The child continued to sleep restlessly with an open mouth. There were also several attacks of otitis media, one of them necessitating a paracentesis, last year.

During the office examination, even without anesthesia, two tremendous folds could be seen jutting out from behind the elevated soft palate. These folds tended to meet in the midline during gagging. The diagnosis was obstruction of the nasal breathing caused by hypertrophy of the salpingopalatine folds.

At operation, only a small amount of lymphoid tissue was found on the posterior pharyngeal wall, in the region of the previous adenoidectomy, but the salpingopalatine and salpingonasal folds were huge. They seemed to start well above the tubal ostium and continued onto the soft palate in a curved line. They ended on the dorsal surface of the palatopharyngeal folds. The weight of the removed plicae was 1 gm. The weight of the residual adenoid tissue found on the posterior pharyngeal wall was only 0.5 gm.

Following this operation, the child was able to breathe through his nose without any difficulty.

In seven of the cases the folds were less than 5 mm. wide but so thickened that it was deemed advisable to remove these masses to prevent any possibility of focal infection.

The interference was not always caused by the enlargement of the salpingopalatine or salpingonasal folds. In four of our cases, the salpingopharyngeal fold caused similar symptoms. In the first case (No. 39), the tonsils and adenoids had been very satisfactorily removed three years previously. The child was six years old when seen in consultation. According to his mother, neither before nor after adenoidectomy could the child breathe through his nose.

Office examination revealed a fold visible on each side of the pharynx; this was accentuated by spontaneous elevation of the palate while the tongue was being depressed.

During the operation, these folds were seen to originate behind the tubal ostium and to continue downward onto the posterior pharyngeal wall. Thus they were the hypertrophied salpingopharyngeal folds. Above they were 12 mm. wide and at the level of the base of the uvula, 7 mm. wide. They were

removed by tonsil punch. The removed tissue weighed 1.5 gm. The salpingopalatine fold was clearly visible anterior to the above described plica. It was relatively small and was not removed.

After operation the child had no more difficulty in nasal respiration.

In most cases it was found difficult to separate the salpingopharyngeal fold from the pharyngeal tonsil during operation. The adenotome or curette catches it together with the adenoids and removes that part of the fold which is on the posterior wall.

In the second and third of these cases of hypertrophied salpingopharyngeal folds, these plicae, although intimately in contact with the mass of adenoid tissue, somehow evaded the adenotome. In both cases the fold started on the torus but ended on the lateral wall of the nasopharynx. It frequently ends on the posterior wall. For this reason it could not be removed with the LaForce adenotome. In one of these two cases (No. 65), the fold was 10 mm. above and 6 mm. in the lower part.

The third case (No. 177) showed the greatest hypertrophy in our series. It was 18 mm. in its upper part and 10 mm. in its lower part.

INDICATIONS FOR COMPLETE ADENOIDECTOMY.

There are three indications for the removal of these folds: the first is to eliminate any obstruction to nasal breathing. Whether the folds form obstruction or not depends upon the size of the choanae. In young children the choanae are narrow; therefore, even a moderate enlargement of the salpingopalatine or salpingonasal folds could obstruct them. Inspection of the choanae by retronasal mirror is of assistance in the diagnosis of such obstruction, and can be performed if, as sometimes happens, the bleeding stops promptly. In Case 93

the salpingopalatine fold was only 5 mm. wide; nevertheless, the mirror examination revealed that it completely covered one of the choanae.

Since then, as a preliminary working rule, all salpingopalatine or salpingonasal folds of 5 mm. or more in width have been removed.

The second indication for removal is that a large mass of lymphoid tissue in the air passages is an excellent nidus for focal infection. Many of the bacteria carried into the nose by the air are deposited in the film of mucus covering the nasal mucous membrane. This film is moved in the pharynx by the nasal ciliated epithelium. The folds which must be passed constitute a barrier, for often they are covered not by ciliated but by squamous epithelium which does not participate actively in moving the germ-laden film onward into the nasopharynx; therefore, an attempt was made to remove every sizable mass of lymphoid tissue, even though it was not obstructive.

The third indication for removal is that in many cases the folds covered the opening of the Eustachian tube and interfered with the necessary equalization of the air pressure between the tympanic cavity and the pharynx, thus leading toward progressive deafness. They also predisposed toward purulent otitis media, because even in cases of mild infection they prevented easy elimination of the contents of the middle ear through the tubal ostium.

TECHNIQUE OF THE COMPLETE ADENOIDECTOMY.

The basic problem in complete adenoidectomy is to remove all macroscopically visible lymphoid tissue from the nasopharynx *under visual control.* The first step is the removal of the tonsils. This gives the operator more space. Before touching the adenoids, the soft palate is elevated. Any retractor is satisfactory, but the modified Love retractor is preferred because it is light and keeps the hand out of the illuminated territory.

We can see the mass of adenoids filling the space between the posterior wall of the pharynx and the soft palate, but we cannot distinguish the salpingopalatine or salpingopharyngeal folds in this big mass of lymphoid tissue, except when the folds are very bulky.

By pushing the adenoid mass backwards with a curved instrument we can find the salpingopalatine fold easily, even though it is small. A metal curved Eustachian catheter is very useful for this manipulation. If we so separate the soft palate from the adenoid mass, we can observe the salpingopalatine fold on the dorsal surface of the elevated palate as a thin or thick, rugged, uneven fold. If it is large, it is sometimes flattened medially, covering the anterior surface of the pharyngeal tonsil. Sometimes it is flattened laterally, covering the tubal opening.

The next step is the removal of the adenoid mass by the LaForce adenotome. It should be sharp so that one can cut, not tear, out the lymphoid tissue. Tearing results in an uneven, ragged wound, making hemostasis more difficult.

The soft palate is elevated and we usually find that a part of the adenoids, especially on the sides, has not been removed. This can be taken out with LaForce adenotome or with a sharp tonsil punch.

Using suction to clear away the blood, we make careful observations and measure the size of the folds. If the bleeding stops quickly, we use a mirror to examine the relative size of the folds and the choanae.

The next step is of utmost importance. The tubal opening must be located. Not once could it be recognized with the unaided eye, for it is hidden among the abundant lymphoid tissue on the lateral wall. The Eustachian catheter solves this problem. With it inserted into the tubal ostium, the important landmarks can be located easily.

Anterior to the catheter lies the salpingopalatine fold; above, the salpingonasal fold; and behind, the salpingopharyngeal fold and the Rosenmüller fossa. All folds originate from

the tube, but the salpingopalatine folds end on the soft palate. The salpingopharyngeal folds end on the dorsal or lateral pharyngeal wall. The usual findings are the salpingonasal and salpingopalatine folds forming a continuous arc, beginning at the fornix and continuing downward on the soft palate, to end most frequently on the dorsal surface of the palatopharyngeal fold. The salpingopharyngeal fold is usually not visible. It is located on the posterior pharyngeal wall and falls into the mouth of the LaForce adenotome together with the adenoids. The lateral pharyngeal wall is often covered with numerous small nodules situated between the salpingopalatine and salpingopharyngeal folds (Barnhill[8]).

Having located the three folds, we remove them if they are too bulky. The most satisfactory instrument seems to be a very sharp conchotome or tonsil punch (No. 2). This instrument can be manipulated with one hand; the other hand is free to lift the palate or to use suction to remove the blood.

This is a great advantage when operating in a deep cavity like the nasopharynx. It eliminates an assistant who has to handle instruments blindly, because the head of the surgeon is blocking the view of the surgical field.

The tonsil punch is used not only for cutting but also for prolongation of the touch sense of the fingers. With it one can feel the hard cartilage below the mucosa; so that the lymphoid tissue can be removed without injuring the cartilage below the salpingonasal fold or the Rosenmüller fossa. Once it has been ascertained by visual examination that everything necessary has been removed, we release the soft palate to its former position.

At this stage of the operation we do not put gauze packings into the nasopharynx. A packing would lift the soft palate and thus elongate the tensor and levator veli palati and put its fibres on tension. The veins which cross these fibres are thus compressed, damming up the blood in the venous capillaries, which cannot collapse, and this in turn tends to prolong bleeding.

The bleeding usually stops spontaneously without packing, especially if we place the head in the natural somewhat flexed position by removing the pillows which are often put under the shoulders of the patient. If the mouth gag was too far open, we partially close it. Both the overextended neck and the too greatly opened jaw exert torsion and pressure on the veins of the neck and so may prevent the free downward flow of blood in the vessels.

We now pause to examine the tonsillar wound and, if necessary, apply ties to the bleeding vessels.

After waiting four or five minutes, we look over the adenoid territory for bleeding points. We do not lift the palate unless it is absolutely necessary, for this invariably causes more profuse bleeding. We simply put the end of the Love retractor into the pharynx until it touches the posterior pharyngeal wall and with the retractor push the soft palate up toward the fornix. We never let the retractor lose contact with the posterior pharyngeal wall. In this way we gain a satisfactory exposure of the posterior wall of the epipharynx and a good part of the fornix. At the same time, the palate and pharynx are pressed against each other, and this pressure aids in stopping the bleeding. The blood is now very carefully removed by cotton pledgets. If any bleeding points are seen, we control them with a modified Coakley slip knot tie.[2]

Ties are used very freely. In fact about the same number of ties are applied in the pharyngeal wound as in the tonsillar fossae. Not once were ties needed in the regions of the removed folds. This may be attributed to two things: first, the tissues were not torn but removed by a sharp cutting tonsil punch; second, the palate was not elevated unnecessarily during hemostasis.

The actual removal of the adenoids and folds never took longer than five or six minutes, not including the time required for hemostasis.

The examination and removal of the folds results in the performance of a more perfect adenoidectomy. During the

exploration of the salpingonasal fold, it is necessary to examine the nasopharynx almost to the posterior end of the choanae. In about 10 of our cases it was found, after a presumably complete removal of the pharyngeal tonsil, that the anterior part of the adenoid mass had not been entirely removed. It was pendulous and hung down from the frontal end of the fornix like a separate lobe, with no apparent connection to the lateral or posterior pharyngeal wall.

According to Testut[7] there are three kinds of nasopharynx:

In the rhinopharynx *a voute ogivale,* the fornix is not in the horizontal plane but looks like a steep roof. From the choanae as its starting point it follows a sharp downward direction, so that when it reaches the posterior wall the angle formed by the fornix and posterior wall of the nasopharynx is an obtuse angle. The nasopharynx is high. This is the most common form in adults (see Figs. 1, 3, 5, 16, 17, 18, 20, 21).

Rhinopharynx *a voute cintre* is built like a flat topped shed. The fornix is the horizontal flat roof. The angle between the fornix and the posterior wall of the nasopharynx is about 90 degrees (see Figs. 2, 3, 8, 9, 10, 13, 14, 15, 19).

About one-third of all the cases show this form; and in these cases one may very easily leave a piece of adenoid tissue anteriorly if he does not go forward far enough with the instruments. In these cases the nasopharynx is deep and makes the examination of the salpingonasal fold more difficult than in the other two forms.

The third form is the rhinopharynx *a voute surbaisse.* This is very common in children. The fornix is at the same level as the lower turbinate and the tube is at the level of the palate. In this form the exploration of the above-mentioned structures is easier. Fortunately this is the kind of epipharynx with which we have to deal in most cases. As the illustrations show, the three different forms of the epipharynx are not sharply divided.

HISTOPATHOLOGIC FINDINGS.

All plica tissue removed was sent to the laboratory where it was described, weighed and where paraffin sections were made of representative portions. In the early stages, portions of adenoid tissue were similarly treated. All three folds present in the main, the same basic elements, *i.e.*, lymphoid tissue and glands covered by respiratory epithelium which often shows squamous metaplasia.

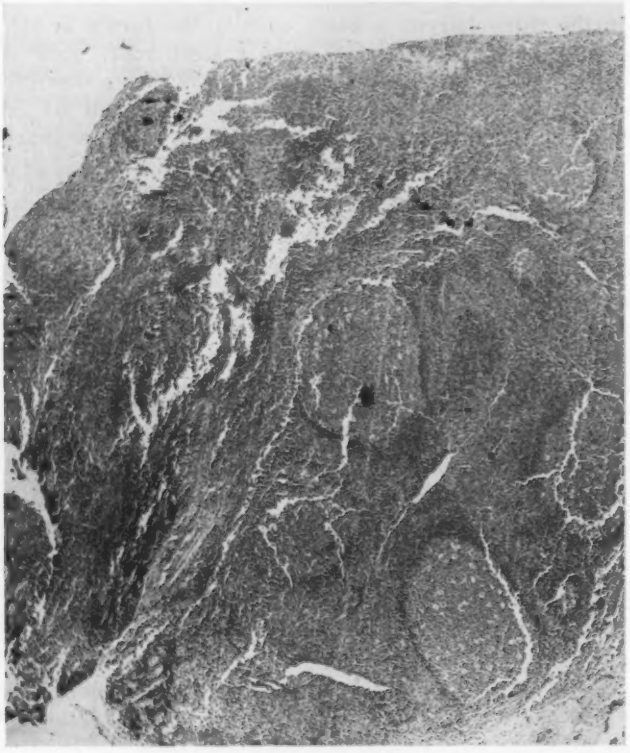


Fig. 1. Plica salpingopharyngea from radical adenectomy. Part of the plica salpingopharyngea. It demonstrates fibrosis, reticulum hyperplasia and the infiltration of squamous epithelium by leucocytes.

Histologically, the folds are quite similar to the pharyngeal tonsil at its inferior border. Usually, the major portion of the plica consists of lymphoid aggregates occupying the lamina propria. Subjacent to this are variable numbers of seromucinous glands, separated from the lymphoid tissue by a thin fibroelastic membrane. There is variation in the surface epithelium: some of the specimens bearing squamous epithelium, others being covered by ciliated columnar cells. Epithelial

Fig. 2. Plica salpingopalatina from radical adenectomy. Plica salpingopalatina presenting glands and lymphoid tissue and predominantly respiratory epithelium with inflammatory infiltration.

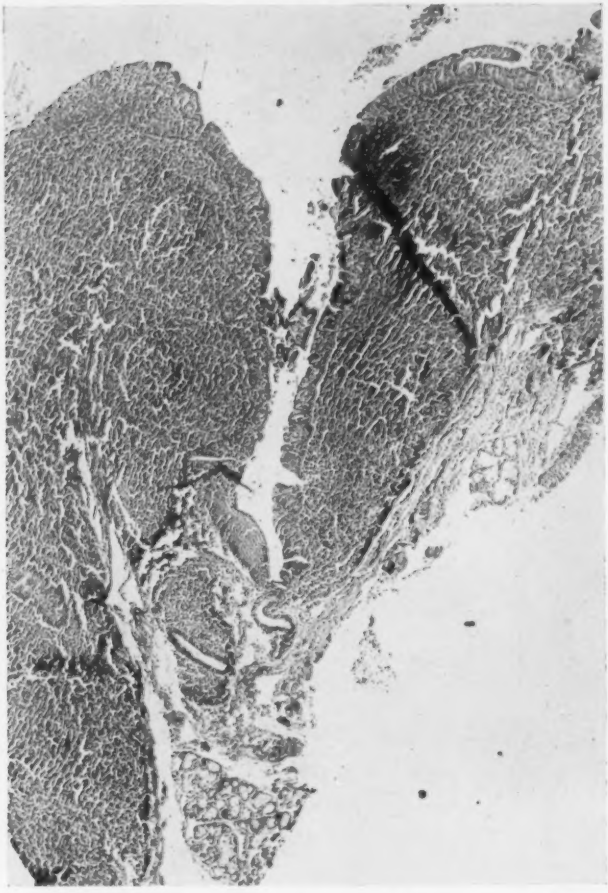


Fig. 3. Plica salpingonasalis from radical adenectomy. Plica salpingonasalis largely lymphoid tissue, with small portion of glandular elements. Squamous metaplasia seen with a duct.

infoldings, which on serial section are seen to be continuous with the gland ducts, give a rugged, slightly nodular appearance to the surface of the folds.

As might be anticipated, none of the specimens presented any acute inflammation. Infrequently, histological evidence of low-grade inflammatory reaction such as focal squamous metaplasia, edema and lymphocytic infiltration of the epithelium, reticulum hyperplasia in the lymphoid aggregates and, rarely, fibrosis were noted. The majority of sections revealed not even this much inflammatory alteration.

In the latter specimens neither voluntary muscle nor cartilage was found, although in the earlier specimens, due to inexperience with the problem, some striated muscle and, once, cartilage, was seen.

CONCLUSIONS.

The literature is replete with articles advising irradiation after adenoidectomy for destruction of the remaining or reformed lymphoid tissue.

In our opinion, many a radium treatment will not be necessary if we remove the hypertrophied folds during adenoidectomy.

It is not at all a rare occurrence that a simple adenoidectomy does not relieve mouth breathing.

We found in our selected series that in 10 cases the enlargement of these folds was so big that the removal was advisable. In two of our cases simple adenoidectomy had been done previously without restoring the nasal breathing. During a subsequent operation no other obstruction was found except for hypertrophied folds originating from the tubal ostium. The removal of these folds restored the free passage of air through the nose.

The elimination of big masses of lymphoid accumulations in a place so openly exposed to bacteria will diminish the number of focal infections.

In this article a radical adenoidectomy is described. This surgical procedure allows the removal of all macroscopically visible lymphoid tissue from the nasopharynx *under visual control.*

SUMMARY.

It is suggested that no adenoidectomy should be considered complete without the examination for removal of diseased or hypertrophied salpingonasal, salpingopalatine and salpingopharyngeal folds.

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LARYNGEAL GRANULOMA FOLLOWING INTRATRACHEAL ANESTHESIA. A REPORT OF FOUR CASES.*

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With the rapid advances being made in surgery, especially relative to the head, neck and thorax, the advantages of endotracheal anesthesia are becoming more and more manifest. Highly skilled surgical and anesthesia teams are of necessity using this form of anesthesia with increasing frequency.

A relatively rare yet significant sequel of prolonged endotracheal anesthesia is nonspecific contact granuloma of the larynx. A review of the available literature reveals that only some 21 cases have been reported since R. J. Clausen's original contribution in 1932.[1] Undoubtedly, many more granulomata have been observed at various clinics but have not been published to date.

A review of the literature, including our four cases, can best be presented in chart form.

In all instances reported, the duration of anesthesia varied from 55 minutes to five hours, with the great majority ranging from two to three hours. Most of these intubations were performed through a laryngoscope by the anesthetist who noted no existing abnormality. Hoarseness, which is the major complaint referable to granuloma of the vocal cord, appeared as early as 10 days and as late as seven months following operation; however, this symptom became apparent in the majority of cases in one to two months after the intubation. Late symptoms may be aphonia and occasionally dyspnea. In 13 instances, the lesion was unilateral and in 12 it

*From the Otolaryngology Service of Dr. Max L. Som.

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was bilateral. On nine occasions the granuloma recurred after laryngoscopic removal.

We believe that the most important factor which may be contributory to the formation of laryngeal granulomata is injury to the mucous membranes. It is only at this anatomical location that these lesions have been described. This trauma to the vocal cords may be of direct or indirect type. The direct type of trauma referred to is that occurring during the actual process of intubation by the anesthetist. This may occur in either the blind or the visual method of insertion of the endotracheal tube. Indirect trauma is that type of injury to the laryngeal mucosa which is caused either by static pressure of the tube *in situ* or by motion of the tube secondary to movement of the larynx and tracheobronchial tree during respiration, coughing, straining, etc. The posterior commissure and vocal processes are the areas where the tube would naturally tend to rest and exert the maximum pressure. The vocal process of the arytenoid is particularly vulnerable to trauma since the cartilage is notably less yielding than muscle and fibrous tissue. Dwyer *et al.*[12] noted that the conventional Magill tube is curved in only one plane. When the tube is inserted into the trachea, the larynx acts as a fulcrum so that the posterior portion of the larynx and the anterior tracheal wall are exposed to constant pressure and trauma. His studies showed more severe congestion of the posterior third of the cords, especially when a large endotracheal tube was used.

It is believed that the initial lesion is a superficial ulceration of the mucous membrane overlying the cartilaginous vocal process. Subsequently, in the attempt at healing, granulation tissue is formed from the submucosa. These granulations must then protrude into the ulcer area which is being encircled by a more slowly growing squamous epithelium. When first seen, such lesions are sessile in character and, due to the differential rate of growth of the tissues, constriction of the base of the granulations occurs, giving rise to a pedunculated mass. In the case reported by Clausen,[1] this pinching off process proceeded to such extent as to allow the granuloma to be coughed up by the patient.

Constitutional factors may possibly make the larynx more vulnerable to trauma.[12] Generalized systemic debility, avitaminosis, anemia, alcoholism and the atrophic mucosa of older individuals may be contributing factors for the lowered resistance to injury by the endotracheal tube.

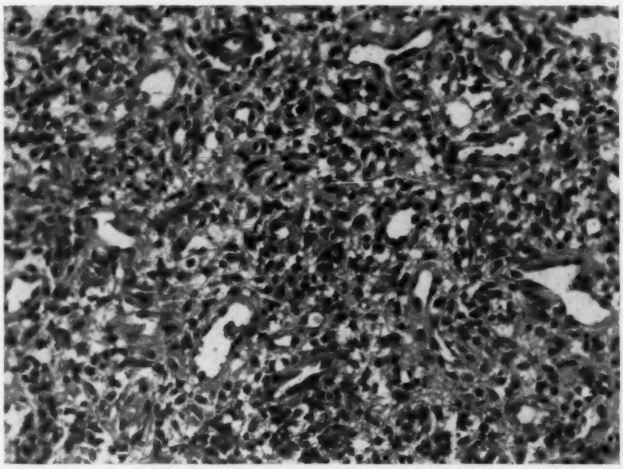
CASE REPORTS.

Case 1: I. C., a 35-year old female, was admitted to the Otolaryngology Service at Beth Israel Hospital on Sept. 20, 1948, with the complaint of hoarseness of three weeks' duration. Mirror examination revealed a rounded pedunculated mass attached to the inferior aspect of the left vocal process. The lesion appeared to be covered by an intact mucous membrane which presented a slightly bluish appearance.

A subtotal thyroidectomy had been performed on this patient about three months previously. Cyclopropane and ether anesthesia was introduced through a nasotracheal Magill tube inserted into the trachea under direct vision. The vocal cords appeared normal at that time. The duration of anesthesia was two hours and 50 minutes.

Postoperatively, hoarseness was noted on the second day and persisted until the sixth day, when it subsided. After a two-month interval, she again developed hoarseness which brought about her admission to the hospital.

Operation: Under direct laryngoscopy with local anesthesia the mass was removed by applying a forceps to its pedicle. The lesion measured about 1 cm. in diameter.



Case 1. Granulation tissue (high power).

Pathology: Granulation tissue with an epithelial surface of stratified squamous epithelium. There were numerous lymphocytes and some polymorphonuclear cells in the submucosal area.

This patient was last seen in January, 1950, about one and one-half years after removal of the granuloma, at which time there was no evidence of recurrence.

Case 2: G. H., a 57-year-old white female, was admitted to the Otolaryngology Service at Beth Israel Hospital on Sept. 19, 1949, with hoarseness of four weeks' duration. Indirect laryngoscopy revealed a pedunculated mass, covered by an intact mucosal surface, arising from the region of the left vocal process.

About seven months prior to this admission, in February, 1949, this patient had undergone a partial colectomy for carcinoma. Endotracheal cyclopropane and ether anesthesia was given throughout a five-hour operation. The vocal cords prior to surgery were normal. There was no postoperative hoarseness.

Operation: Using indirect laryngoscopy with local anesthesia, the mass was removed at its pedicle by a grasping forceps. The tumor measured 8 mm. in diameter and the pathological report was nonspecific granuloma. The patient was again seen on Jan. 9, 1950, at which time she again complained of hoarseness of two weeks' duration. Laryngeal examination revealed a recurrence of the mass over the left vocal process. This tumor was again removed with the indirect laryngeal forceps.

She has been followed in the outpatient department and no evidence of recurrence has been noted to date.

Case 3: M. F., a 70-year-old white male, was admitted to the Otolaryngology Service at the Beth Israel Hospital on Jan. 9, 1950, with the complaints of hoarseness and difficulty in raising sputum, of two weeks' duration. Mirror examination revealed bilateral masses attached to the vocal process of each vocal cord. They were pedunculated and would descend below the cords on inspiration and swing out over the cords on expiration. An intact mucosa covered both tumor masses.

On Nov. 7, 1949, two months previously, a resection of the sigmoid portion of the colon had been performed under endotracheal cyclopropane and ether anesthesia. The intubation was described as being "difficult." No abnormality of the vocal cords was noted preoperatively. Anesthesia lasted two hours and 30 minutes. There was no hoarseness during the postoperative course in the hospital.

Operation: Under indirect laryngoscopy with local anesthesia, both polypoid masses were removed with the laryngeal forceps. Each mass measured about 7 mm. in diameter. The pathological report was nonspecific granuloma.

Postoperatively, this patient has been followed in the outpatient department and showed no evidence of recurrence when last seen on June 14, 1950, five months later.

Case 4: C. C., a 39-year-old white female, was seen in September, 1947, with a history of having had a radical mastectomy performed at another hospital four weeks previously. The duration of the surgery was well over two hours. Anesthesia was endotracheal in type. No complaints were noted in the hospital.

Author	Duration of Anesthesia	Type of Intuoation	Onset Symptoms of Granuloma	Unilateral or Bilateral		Recurrence
Clausen	(1) ?	?	5 mos.	X		no
Gould	(2) 6 hrs. 10 min.	blind	2½ mos.	X		yes 2 mos.
Cohen	(3) 55 min.	blind	2½ mos.	X		no
Smiley	(4) 1 hr. 35 min.	direct, larynx normal	10 days	X		yes 2 mos.
Farrior	(5) 3 hrs. 45 min.	direct, larynx normal	14 days	X		no
Kearney	(6) 5 hrs.	direct, larynx normal	1mo.		X	no
Tuft	(7) 3 hrs. 45 min.	blind	1 mo.		X	no
Barton	(8) 5 hrs.	direct, larynx normal	2½ mox.	X		no
Barton	(8) ?	direct, larynx normal	2 mos.	X		no
Finer	(9) 3 hrs. 40 min.	direct	1 mo.	X		no
Hill	(10) 4 hrs.	direct, larynx normal	1 mo.		X	yes,2 mos.
Wattles	(11) 1 hr. 40 min.	Direct, larynx normal	2½ mos.		X	no
New & Devine	(13) not given	not given	not given	2	7	5 patients
		CASES PRESENTED	IN THIS PAPER			
1. C.	2 hrs. 50 min.	direct, larynx normal	2 mos.	X		no
G. H.	5 hrs.	difficult intubation	7 mos.	X		yes, 4 mos.
M. F.	2 hrs. 25 min.	blind difficult	2 mos.		X	no
C. C.	2½ hours	unknown	4 weeks	X		no

Shortly after discharge from the hospital, the patient became hoarse. Mirror examination revealed a mass arising from the right vocal process, covered by an intact mucosa. The tumor almost completely occluded the interarytenoid space.

Under indirect laryngoscopy with local anesthesia, the mass was removed with a grasping forceps. The pathological report was nonspecific granuloma.

The patient was re-examined in January, 1948, and no evidence of recurrence was noted. The patient has had no laryngeal complaints to date (September, 1950).

Case 4. Low power showing granulation tissue and ulcer base.

DISCUSSION.

From a clinical standpoint, the most significant symptom of this condition was hoarseness occurring from one to two months after endotracheal anesthesia. The importance of early diagnosis and prompt removel of the lesion must be stressed. In our review of the literature several cases were noted where treatment was delayed from two to four weeks, and the patients subsequently showed aphonia and marked dyspnea, even to the point of impending asphyxia. These cases were reported by Kearney,[6] Gould[2] and Smiley.[4]

The single curve of the Magill tube, as pointed out by Dwyer *et al.*,[12] may be more of a contributing factor in the etiology of these lesions than is commonly suspected. The

modification of the Magill tube to a double curve, simulating the anatomical curvatures as suggested by Dwyer *et al.*, is certainly worthy of consideration as a possible method of preventing these lesions. It would seem that the endotracheal tube should be of the smallest size compatible with the lumen of the larynx.

SUMMARY.

1. A review of the literature reveals 21 cases of granuloma of the larynx following endotracheal anesthesia.

2. Four additional cases from our clinic are discussed.

3. The etiological factors, pathology, and possible methods of preventing these lesions are also presented.

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77 Park Avenue.

UNIVERSITY OF ILLINOIS INITIATES MODIFIED PROGRAM.

For the period of the national emergency, the University of Illinois College of Medicine will offer a combined three-year residency training program in otolaryngology, which will include the basic course material, in fulfillment of board requirements.

Residents will rotate through the Research and Educational Hospitals, the Illinois Eye and Ear Infirmary, the Hines Veterans Administration Hospital and the various affiliated institutions. The residency will be so flexible that should it be interrupted because of military service, the period of training may be resumed upon returning to civilian life.

Under this arrangement, no course fee is to be involved, and in the case of most of the aforementioned institutions, a stipend is provided.

For further information, kindly address: Head of the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Ill.

CORRECTION.

In the October, 1950, issue of THE LARYNGOSCOPE, page 1053, an article entitled, "New Hearing Aid Features World's Tiniest Receiver — Offers Widest Frequency Range, More Overtones," the word advt. (advertisement) was omitted from the end of this article.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Georgia Society of Ophthalmology and Otolaryngology will hold its annual meeting at the General Oglethorpe Hotel in Savannah, Ga., on March 2 and 3, 1951.

The lecturers and their subjects are: Dr. A. D. Ruedemann, Detroit, Mich., Cataracts as a Medical Problem and Selection of the Type of Operation in Cataract Surgery; Dr. Peter C. Kronfeld, Chicago, Ill., Diagnosis and Medical Treatment of the Glaucomas and the Surgical Treatment of the Glaucomas; Dr. William C. Owens, Baltimore, Md., Surgical Treatment of Horizontal Muscle Deviations and Surgical Treatment of Vertical Muscle Deviations; Dr. C. Stewart Nash, Rochester, N. Y., Functional Diseases of the Nose and Otolaryngological Mishaps; Dr. Philip Meltzer, Boston, Mass., Simple, Practical Therapeutic Measures in Otologic Practice — Surgical and Non-Surgical, and The Conservation of Hearing in Chronic Suppurative Otitis Media; Dr. Edwin C. Broyles, Baltimore, Md., Tumors of the Larynx — Benign and Malignant — and Summary of Otolaryngological Conditions That Require Expert Attention.

Members and guests are invited to make their reservations directly with the hotel. Registration fee for the lectures is $20.00.

In Memoriam

PERRY GLADSTONE GOLDSMITH, C.B.E., F.R.C.S., M.D., Ch.M.,

1872-1951.

In the passing of Dr. Goldsmith on Jan. 21, 1951, Canada lost perhaps her most distinguished representative of the past half century in the specialty of otolaryngology. As a staunch supporter of the British school, he formed one of the strongest links between this group and that of America, as he always took an active interest in all the senior American societies.

He was born in the small Ontario town of Warkworth, as the son of a medical practitioner and graduated from Trinity College of the University of Toronto. His postgraduate training in otolaryngology was taken in England, where he was house surgeon to such great names as Mr. Dundas Grant and Sir Sinclair Thomson, both of whom he frequently quoted. Before World War I he was attached to the University of Toronto and the Toronto General Hospital, but, as he was actively interested in army affairs, he enlisted and went overseas with the First Canadian Division as a battalion medical officer in 1914. His army career was distinguished throughout and he was the commander of the first Canadian field ambulance sent to France. He had successive posts as commanding officer of various units and was "mentioned in dispatches" in two successive years. In 1918 he was awarded "Commander of the British Empire" (military division), which is a coveted distinction.

Following the war, Dr. Goldsmith resumed his civilian duties and, in 1922, was appointed professor of otolaryngology in the University of Toronto and chief of service of this department in the Toronto General Hospital. He ably filled

these appointments until 1939, when he retired to continue private practice until 1950.

Perhap his most useful service to our specialty was his presence as an examiner, for a good many years, on the American Board of Otolaryngology. His wise counsel and his sympathy towards the candidates was an example which was appreciated by all his more junior confrérès. He will be missed from his position as senior counselor on this board.

In addition to being a Fellow of the Royal Society of Medicine of England, he was also a Fellow of all the senior American societies. These included: American Otological Society; American Laryngological Association; American Laryngological, Rhinological and Otological Society, Inc.; American Academy of Ophthalmology and Otolaryngology; American College of Surgeons. He was president of the American Laryngological, Rhinological and Otological Society, Inc., when it met in Toronto, in 1934, and was guest of honor on one occasion of the American Academy of Ophthalmology and Otolaryngology.

It is difficult not to add a personal note to this obituary. Dr. Goldsmith was one of the most ardent readers of the English and American literature of our specialty and also that of general medicine and surgery. He was kindly, helpful and critical of the juniors in his department. An ardent gardener, his rock garden was noted as one of the most outstanding in Canada and he was elected a life member of the Ontario Horticultural Society. His love of the arts was well illustrated by the splendid collection of paintings, china and antique furniture so profusely found in his home. These all showed evidence of a soul much more deeply rooted than that of the average physician or surgeon of our day.

At this point I quote from two letters of appreciation:

"Another tall pine has gone.

"Goldsmith, like Birkett, was a link with old times, and, like him, was a good soldier.

"I used to have good times with him going out to various rock garden specialists. We had a nice trip to Portland gardens when he was last here. He was a real dirt gardener.

"We shall not soon forget his magnificent dinner at the Royal York with haggis and all the panoply of pipers playing. He reached his apogee that night." R. A. F.

* * * *

"Dr. Goldsmith had his first training as a general practitioner and never lost the broad medical perspective which this gave him. Often the loss of perspective is the chief sin of specialism.

"He continually warned his patients not to consider a slight inconvenience a major complaint. He once said, partly as a joke, that this was a characteristic trait of the American patient.

"I have been told that students never cut his lectures because they were meaty, practical and bright." H. P. M.

* * * *

The funeral services presented some sadness in that he had been predeceased by his wife and only son so that he left only a sister and a granddaughter behind him. The honorary pallbearers were represented by three emeritus professors from various departments and three active professors. The American Board was represented in this group by Dr. Harris P. Mosher, who had been a personal friend for many years, and by Dr. Percy E. Ireland. It speaks well for the respect in which he was held that such a distinguished group of medical confrérès were present to pay honor at his last rites.

P. E. I.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

Feb. 1, 1951.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model 45.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; tarion Model E-4; Otarion Models F-1 and F-2.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

(See Micronic.)

Silvertone Model 103BM.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model M-35.

Manufacturer: Microme Co., 727 Atlantic Ave., Boston 11, Mass.

Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model P-15.

Manufacturer: W. E. Johnston Mfg. Co., 708W. 40th St., Minneapolis, Minn.

Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.
Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 1700.
Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.
Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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BASIC DESIGN CHANGE MINIMIZES CASE NOISE IN NEWEST MAICO INSTRUMENT.

One of the most common patient objections to hearing aids is "case noise" caused by the rubbing of the wearer's clothing against the acoustically sensitive surface of the instrument's microphone.

Watson and Tolan find that, of various "extraneous noises," "case noise" is generally the worst offender.

Silverman and Taylor advise: "If the instrument has any great internal noise, either electrical or from friction against your clothing you will (score) a black mark against the instrument."

Now, for the first time in a hearing instrument, the problem of clothes-rub noise has been approached by a basic design change in the positioning of the microphone in the transmitter. In this new type hearing aid, the Maico *Top Secret*, the microphone is top-mounted and recessed in the transmitter unit, so that clothing does not make contact with the sound-live surface. Patients can anticipate new fidelity of hearing, unmarred by the scratching and crackling noises that until now have often characterized the use of electronic hearing aids.

In building this advanced instrument, Maico engineers did not sacrifice other desirable design features — for example, small size. The new Top Secret instrument is only two-thirds the size of a standard cigarette pack, a fact of importance to hearing aid wearers who, for reasons well understood by doctors familiar with the psychology of the deafened, almost always prefer an instrument that can be easily concealed in a pocket or beneath the clothing; nor has there been any sacrifice in such vital factors as amplification, frequency response, or tone quality.

(advt)

SONOTONE PERFECTS WORLD'S MOST POWERFUL HEARING AID.

This most powerful wearable hearing aid, the first six-tube instrument in history, has been introduced by Sonotone Corporation. Despite its unprecedented power, the compact Sonotone "966" is as wearable as Sonotone's previous miniature aids.

The "966" was especially designed to assist patients with extreme hearing impairments. Its six tubes, double the number usually used in other hearing aids today, generate sufficient power to bring hearing to thousands of deafened persons who could not be helped by any other instrument now available. The '966" can overcome most hearing losses up to 100 decibels, and may be adjusted to other severe losses in which there may be only faint audiometric indications of residual hearing.

The new Sonotone instrument is especially suitable for bone conduction, the method of hearing on which thousands of hard-of-hearing persons depend. This new six-tube instrument revitalizes bone conduction as the most satisfactory way of hearing for many persons. Wearing of bone-conduction hearing aids has now been made easier for many persons through elimination of the customary headband by Sonotone's "Noband," a plastic film.

Incorporated in the Sonotone "966" is a new "Robot Sentry" which suppresses the reproduction of very loud noises which the instrument is capable of handling, yet permitting desirable sounds to come through in proper balance. Another exclusive Sonotone device, an "Electronic Brain," limits battery drain, despite the six tubes, to what is needed at any moment of operation. For most normal conditions of use, the battery power consumed is no greater than that of ordinary hearing aids. Only when maximum power is required by a user are the batteries called upon for full output. Another economy device permits the use of outside longer-lasting batteries, although the instrument is mostly worn as the usual all-in-one with batteries enclosed.

(advt)

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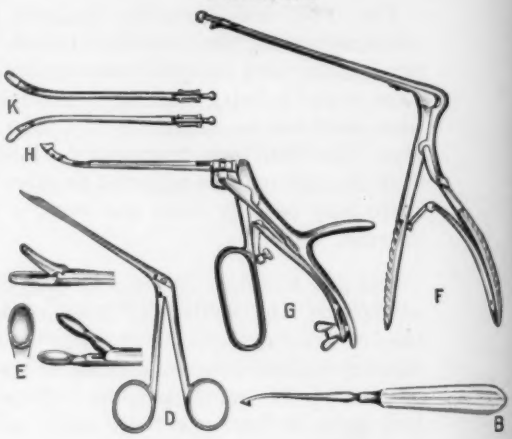

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